ANNALS OF SURGERY

VOL. 105 MAY, 1937 No. 5

TRANSACTIONS
OF THE
SOUTHERN SURGICAL ASSOCIATION

MEETING HELD IN EDGEWATER PARK, MISS.
December 15, 1936

PRESIDENTIAL ADDRESS

WIDER HORIZONS FOR THE SURGEON

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THE generation of American surgeons who were approaching the zenith of their careers when I was a young man getting my training comprised among its leaders many men of a type that has few exemplars among my own contemporaries. Some of these men have left us. Fortunately, many of them are spared to us, still actively at work or filling the important rôle of elder statesmen to our beloved guild of surgeons. They were and are something more than masters of their specialty in the healing art. They possessed a vigor of energy, a gift of expression, an inspirational force as teachers that were characteristic. Furthermore, they were endowed with that quality of magnetism, of personal charm, and dramatic sense that made many of them public figures of renown—what the current phrase describes as "colorful personalities." Among them are such names as Murphy, Ochsner, Joseph Blake, Brewer, Deaver, Matas, the Mayos, Crile and our own Doctor Finney and many more. The mere reading of the list has the bugle-call effect of the names of victories on regimental banners. But when I look for men who may be regarded as successors of the same type and qualities, their scarcity is most striking, and, as a member of a lesser generation, I am inclined to hide my diminished head in humility, if not in shame. Yet, I fully share the optimistic views of the future of surgery that were so forcefully presented to us two years ago in Dr. Frank Boland's presidential address before this Society.

Perhaps this hopefulness, despite our lesser stature than the Great Generation, is not so inconsistent as it seems, for we are told that each generation stands on the shoulders of its predecessors and that an ordinary man standing on the shoulders of a giant can see farther than the giant can. The exploration of this ever widening horizon is the purpose of my paper.

Since the beginning of this century, science in many fields has made revolutionary advances. Physics has broken the "indestructible" elements of our student days into electrons, protons and neutrons. Astronomy has extended the universe beyond the comprehension of the ordinary layman. Organic chemistry has recognized, refined and synthesized a multitude of new products. The geneticists have traced farther and farther the factors that influence heredity. Biology has learned much of the properties of growth and repair of living tissues. Endocrinology and the study of nutrition have opened wide new fields. In many other branches of science, too numerous to mention, great strides forward have been made. What has all this to do with surgery?

Is not surgery an art in which the deft and bold hand works its masterpieces of skill for the benefit of suffering humanity? It is, indeed. Is not surgery a scholarly discipline in which the learning and experience of the past are critically analyzed, correlated and catalogued? It is, without doubt. Is not surgery a high calling—almost a priesthood—in which character, integrity, humanity and devotion to high ideals are the very basis and cornerstone of the structure? It is, most emphatically. Surgery is by universal acclaim all of these things and it is more. It is a vital and integral part of the rapidly growing field of science—the development of man's manifold activities that, perhaps, chiefly characterizes the last three centuries of his history.

The chemist and the physicist, who explore the intimate structure of the atom, work on the simplest known forms of matter to restrict the difficulties of their problems. When they come and report to us their findings about helium and hydrogen, let us not forget that general laws of atomic structure apply also to the atoms that make up the material of the human body. The men who seek to understand the laws governing solutions and gels, colloids and semipermeable membranes, in the comparatively uncomplicated experiments of the laboratory, are working on problems that directly concern the behavior and mechanism of living beings. The students of radiant energy in its various forms are engaged with a subject that touches medicine at innumerable points. If this be true of the so called basic sciences of the nonliving world, what of the many phases of biologic investigations? Much more obviously, the thoughts, the experiments, the discoveries and the conclusions of the biologists are of the greatest importance to those of us who work in the field of medicine—which is, after all, nothing but a specialized field of biology. And let us not forget for a moment that great field of biology that spreads over into philosophy, religion, ethics and psychology—the biology of the mind. If any of us need reminders of the bearing of the mind on health and physical welfare, we have Christian Science as a monumental object lesson.

Lest any one object that I am straying far afield from the proper domain

of the surgeon, let me relate to you certain gleanings from casual reading in various scientific fields during the past year and the chain of ideas that they set going. In a most stimulating address before the Zoologic Section of the American Association for the Advancement of Science, Dr. Oscar Riddle spoke on certain phases of evolution. The part of his address that is pertinent here may be condensed as follows: It is known that large numbers of inorganic compounds which probably could not exist during very early phases of earth's history did come into existence in epochs preceding the appearance of living things. It has also been proved that formaldehyde, sugars and other organic chemical substances, some of them containing nitrogen, are generated under conditions which must have prevailed on the earth's surface before, during and after the origin of living things. The agents to the building up of such organic matter are none other than sunlight, ordinary temperature, colored surfaces, water and carbon dioxide. There exists scarcely a doubt that during long periods of the earth's history, preceding the appearance of living matter, many localized areas of its surface provided suitable conditions for the synthesis of sugar and some amino acids.

There is now a new word to add to this discussion of the approach of nonliving toward living substance. For some years it has been fairly evident that the substances or units known as viruses are smaller than any visible cell or organism and that they, nevertheless, show the one property which is most characteristic of life—that of self-reproduction. They have seemed part of a bridge between those smaller but lifeless organic molecules which, as we have just noted, form spontaneously on the earth's surface, and those larger molecular aggregates which are bacteria and cells endowed with all the properties of life. The virus appears to stand nearer to life than to the spontaneously formed sugars and amino acids. Within recent months Stanley has prepared a crystalline protein which is apparently the virus of the disease of tobacco known as "mosaic." These crystals, of definite chemical and physical properties, unchanged after ten successive recrystallizations, have the remarkable power of indefinitely building themselves anew when brought into contact with suitable living tissue—the tobacco plant—exhibiting thus one of the characteristics of living matter. Now, when the known properties of this virus—its molecular size and its power to reproduce itself and many other qualities—are compared with those of the gene, there is a striking parallel. The genes, be it remembered, are those component units of the chromosomes of living cells to which biologists have attributed many important functions of living matter, notably the carrying of hereditary traits from one generation to another, and the peculiar characteristics of each species. So much for a very inadequate extract from Doctor Riddle's most thought-provoking article.

Recently Rous and his associates of the Rockefeller Institute have published a series of experiments on rabbits with a virus disease. In certain strains of wild cotton-tail rabbits there occurs an endemic infectious form of skin papilloma which is transmissible by a filterable virus. When this virus is transmitted under proper conditions to laboratory rabbits, its reproduces the

papillomatous tumors, and, in a certain number of instances, these ordinarily benign tumors take on definitely malignant changes and become cancers. Doctor Rous and his associates are properly conservative in the conclusions they draw from these experiments, but it seems well proved that the introduction of this agent, the filterable virus, is the primary cause of the development of the cancers.

To summarize: Riddle gives us a long look backward over the earth's history, beginning with simple chemicals and the energy of sunlight and leading up to complicated chemical substances. Stanley tells us of a filterable virus which is a nonliving, crystalline substance but which in structure and behavior suggestively resembles those components of living cells—the genes—that play a large part in inheritance and reproduction of life. Rous establishes a relationship between a virus and a form of malignant disease. At this point I would remind you that a presidential address in this Society is not by custom limited to restrained scientific caution. I am now about to avail myself of this latitude by sailing off "into the blue" of pure imaginative speculation with no excuse except the privilege of doing so and the inviting take-off provided by the serious work referred to above.

Admittedly, the great problem of medicine today is the cancer problem. We know a great deal about cancer but are ignorant of far more. We can do something toward its cure and something more toward its alleviation and restraint, but so pitifully little compared to what we would like to do. It is an object of endless thought, study and experimentation over the whole medical world. Perhaps, even if we knew its cause in all its possible complexity, we should still be unable to solve the problem of its successful treatment, but surely until we do understand its nature better, our attempts at treatment are essentially blind—and unsatisfactory. There have been so many theories advanced to explain the development of malignant disease that another guess can do no particular harm except to expose its author to devastating criticism, and since presidential addresses are not subject to discussion, my chastisement will at least not be immediate and public. To make my offense as brief as possible, it shall be reduced to skeleton outlines.

The most acceptable view of malignant disease at present regards the cancer cell as a new species of cell, essentially a new animal, derived from a presumably normal cell by some form of mutation. This mutation once established, the cancer goes along leading its own life, independent largely of restraint by the host from which it sprang and from which it derives food, shelter, warmth and protection, but to which it returns no service, not even physiologic obedience. It is reasonable to suppose that this change occurs because of some alteration in the laws of inheritance which normally provide that daughter cells shall develop along established lines, subject to the physiologic restraints that affect the mother cell. The mechanism of inheritance appears to be bound up intimately with the genes. Any agency, external or internal, that violently altered the internal structure of the cell, would undoubtedly kill it. But an agency that modified the cell in a less extreme

manner might conceivably result in a cell that could still live but was so changed that it no longer was a legitimate descendant of its parents. Such an agency might conceivably be a chemical body so similar to the genes themselves that it might supplant a gene during the process of cell division, or be incorporated as an extra gene, or combine with a gene to form an abnormal gene. If a filterable virus, with the properties that have been attributed to them, were available at opportune moments during cell activities, especially division, it is not an unimaginable fancy that some such action might take place. Again, if radiant energy in some form, with its known power to alter molecular arrangements, were to disturb, slightly, the chemical composition of the large molecules in the cell nucleus, a similar effect might result. Or if chemical substances like those in certain coal-tar derivatives, of known resemblance to the hormones of the body, were to enter into combination with the substance of the cell so as to alter or damage the mechanism of inheritance, a series of abnormal daughter cells might be inaugurated. Now, it is known that all three of these particular agencies may actually produce such results, and perhaps many other agencies as yet less clearly recognized may have similar effects. This speculation as to the manner of action of these agencies is not, perhaps, of practical value at present, perhaps not even very new or original, but it may at least have the merit of crudely visualizing a process that interests all of us intensely and of stringing together some of the known facts, even though the thread on which they are strung is very tenuous.

I seem to have gotten rather far from my subject in this excursion into the realms of fancy, yet this diversion may serve as an illustration of what I am trying to express. If the men who are working with viruses of plant diseases have made important advances, we should know about it, not merely as a matter of general cultural or scientific interest but because it may throw light upon some surgical problem. When the metallurgists produce new alloys or the students of plastic materials evolve substances with new properties, these things may be especially adapted to some surgical purpose as instruments or braces. When the cellulose chemists develop new threads or fabrics, they may be useful for dressings or sutures. Do any of you feel that we yet have the ideal suture material? The students of electricity, in its manifold forms, have indirectly contributed enormously to diagnosis and therapy in all fields of medicine, and there is no reason to believe that they have exhausted the possibilities, but we must be alert to recognize and utilize the aid they make available. To extend similar comments to the many diversified fields of all the sciences is unnecessary. But it should not be supposed that science alone is of concern to us. Today as never before we are affected by matters economic, social and political, and surgery, like every other human activity, does not escape these influences, which are real and practical in everyday experience.

But you say I am asking impossibilities. Surgery itself is a demanding mistress, taking all the energy, the time, the thought that any man can give. How then expect the surgeon to keep abreast of modern physics—a subject so

abstruse that the average man is helpless at the thought of it; and chemistry with its multitudinous subdivisions, each actively and vigorously developing, what can we hope to do with that? Even our near relative, biology, is so vast that our part of it seems a little corner. The answer is, of course, that no one of us can even remotely approximate the knowledge that we should like to have and that would be so helpful to us. The day of the universal scholar, who took all learning for his province, is long since past. But what no one man can do, many men acting together may accomplish. Fortunately, we are united in an organization to combat the forces of ill health and are each component cells in a highly developed whole, not isolated amebae leading solitary lives. However, if the organization is to function properly, each unit must perform his own full duty and one of those duties is to stretch his mental horizon beyond the narrow scope or his daily task. Let the man who has a mechanical bent learn from the engineers and mechanics how to devise more useful tools for the needs of surgery. Let the man with a physiologic trend help us to improve the preoperative and postoperative care of our patients. Let him with chemical leanings give us better antiseptics and anesthetics. We need to know what the bacteriologists have to tell us about immunity and serums and vaccines. We need so many things that each can help. And most of all, we need the large view, the wide horizon; not only facts and knowledge gathered from many fields, but the sense of proportion, of balance, of sanity, that comes from seeing life, surgery and ourselves in true relations and proper setting. And lastly we must have a forum for the exchange of our gleanings from far and near. The various organizations of the profession provide such forums, and we are gathered tonight in one such meeting place, that I hold to be second to none in its many useful and charming qualities. If we would make it still better, richer for each of us both in practical matters and in the growth of our souls, let us send our minds to wander the far horizons and bring back here the fruits of our wanderings. It is said that in the heyday of Venice, each of her many vessels that sailed all the known seas had to bring back some treasure for the enrichment of St. Mark's Cathedral. St. Mark's, in consequence, remains today one of the world's wonders. The Southern Surgical Society may well become the St. Mark's of the surgical world.

SURGICAL TREATMENT OF FACIAL SPASM

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PAROXYSMAL disturbances of function are characteristic expressions of the surgical diseases which involve the cranial nerves. The well known paroxysms of pain of tic douloureux of the trigeminal and glossopharyngeal nerves and the paroxysmal vertigo of the Ménière's syndrome strikingly illustrate the tendency of these nerves to manifest disturbance in an explosive manner. The facial and spinal accessory nerves likewise may be involved in paroxysmal affections which, by exaggeration of motor function, produce both deformity and disability.

Surgery of the facial nerve may be required to restore its function when the nerve is paralyzed and to reduce or abolish its function when spasm of the muscles it supplies causes a disabling facial deformity. Facial spasm may be unilateral or bilateral and either clonic or tonic in type. In severe cases it not only causes an embarrassing deformity but because of interference with vision in bilateral cases it may result in serious disability.

The pathology of facial spasm is entirely speculative. Wilfred Harris[1] believes that clonic unilateral facial spasm is due to a degenerative lesion of the nerve at or below the geniculate ganglion. In support of this opinion he cites the occurrence of spasm following recovery from facial paralysis due to injury of the nerve trunk. Harris rejects Gibson's[2] theory that facial spasm results from pathology similar to that of spasmodic torticollis on the grounds that in the former condition only one nerve (the facial) is involved, while in the latter disorder the cervical nerves as well as the spinal accessory are implicated. In further support of the neural basis of clonic unilateral facial spasm may be mentioned the tendency of the affected facial muscles in long standing cases to develop weakness and contractures. Bilateral facial spasm is most probably of cerebral origin and may result from encephalitis. This appeared to be the cause in one of the patients under consideration in this paper.

Parker[3] reports two cases of bilateral facial spasm (paraspasme Sicard). Both cases were believed to be due to cerebral disease. One had definite Parkinson's disease and the other had an early Parkinson's syndrome following encephalitis. There is evidence to support the opinion that the pathology of bilateral spasm differs from that of the unilateral type. The muscle contraction of the former is tonic in character while the latter is clonic. Moreover, in bilateral spasm there is often spasm of other groups of muscles affecting phonation and deglutition. Evidence is lacking to support the view that either the bilateral or unilateral spasm is due to a lesion of the cortical centers controlling facial movements.

Spontaneous recovery from well developed facial spasm probably never occurs. Starting usually as blepharospasm, the contractions spread until they involve the musculature of one or both sides of the face and extend into one or both platysma muscles. Bodily movements, excitement or fatigue increase the spasm while quiet and tranquillity give some relief. The contraction of the facial muscles, particularly those about the eye and angle of the mouth, produces a conspicuous deformity.

The victims of the severe types of bilateral facial spasm (paraspasme Sicard) are often completely disabled. They are unable to walk safely in traffic, unable to read, and sometimes because of closure of the eyelids and contraction of the muscles about the mouth it is with great difficulty that they can feed themselves. The expression of the face in the severe types of bilateral spasm is similar to that registered after tasting some acrid, pungent substance.

FIG. 1.—Incision for exposure of the trunk and main branches of the facial nerve, the trunk and main branches being shown diagrammatically. Through this incision, which is made under local anesthesia, the trunk can be followed posteriorly to give exposure to the mandibular branch and main trunk of the nerve.

During the past year we have had under observation five patients with facial spasm. There were three patients with clonic unilateral spasm and two in whom the spasm was bilateral and predominantly of the tonic type. The patients with unilateral spasm sought relief chiefly because of the embarrassment resulting from contraction of the facial muscles, particularly those about the eye. Reading was difficult and the patients were continuously disturbed by the facial grimaces. Many kinds of therapy had been tried without relief. The contractions persisted with variations but with increasing intensity and annoyance.

Two of the patients had severe disabling bilateral tonic contractions of the entire facial musculature. The first of these patients was a man, age 27, who probably had encephalitis as a result of chronic manganese poisoning. The deformity in this case was very striking. The whole face was involved in almost continuous tonic contraction which closed the eyes tightly and retracted the angles of the mouth. The voice was impaired and difficulty in talking was intensified as the facial contractions became more severe. The tonic contractions appeared in rapid succession, giving an almost continuous picture of a deformity in motion. The patient was completely disabled and had to be led around.

The second case of bilateral spasm was in a frail man, age 74. In this case there was no history of encephalitis and no evidence of arteriosclerosis in excess of that which might be expected in a man of his age. The spasms

in this patient had started around the left eye two years before and gradually spread until they involved the entire distribution of both facial nerves. The contractions of the platysma muscles forcibly depressed the chin while the tonic spasm of the orbicularis oculi closed the eyelids tightly. The whole face was in almost continuous spasm, producing hideous grimaces which followed each other in rapid succession.

Medical treatment and psychotherapy are of no benefit to facial spasm.

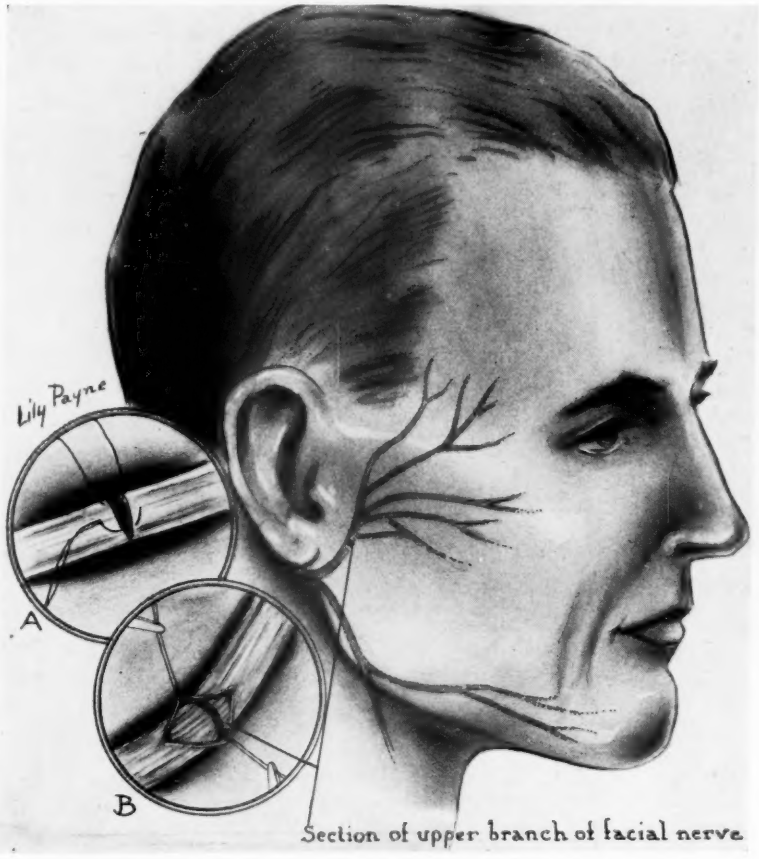

FIG. 2.—Section of the nerve, leaving a segment of the sheath attached as shown in insert A. By using a fine knife in some larger trunks the sheath may be separated and section of the nerve fibers made without division of the sheath, as shown in insert B. When this is possible, no suture is required. Magnification of the nerve in inserts A and B for better illustration. Fine needles and silk sutures are necessary for accurate approximation.

The condition can be relieved only by paralysis of the nerve by section, or by injection of the nerve with alcohol. With recovery from the paralysis the spasm usually returns but the patient is grateful for a period of relief which may last from six to twelve months.

The nerve trunk may be injected with alcohol where it leaves the stylomastoid foramen but this procedure is not so simple as it might appear. Injection of the facial trunk produces paralysis of the entire distribution

of the nerve. Individual branches of the nerve may be injected just at the point where they cross over the ramus of the jaw but the landmarks here are not especially reliable due to the considerable variation in the arrangement of the nerve branches. There is some objection to injection of alcohol in the branches of the nerve inasmuch as alcohol may cause scarring of the nerve and interfere with recovery of muscle function, provided some form of anastomosis is later required.

In unilateral clonic spasm in which the greatest contraction is in the orbicularis oculi muscle group, we have divided the nerve through a short incision, under local anesthesia (Figs. 1 and 2). We have preserved the

FIG. 3.—Male, age 43, with clonic spasm of the right side of the face for one year with most annoying contraction in the orbicularis palpebrarum. The lower branches of the nerve were involved to a lesser extent. Section of the branches to the orbicularis, frontalis and muscles of the upper lip under local anesthesia on December 9, 1936. Photograph shows patient in slight spasm of the right side of the face. At times the eye was forcibly closed by the spasm. There was beginning contracture of the muscles of the affected side.

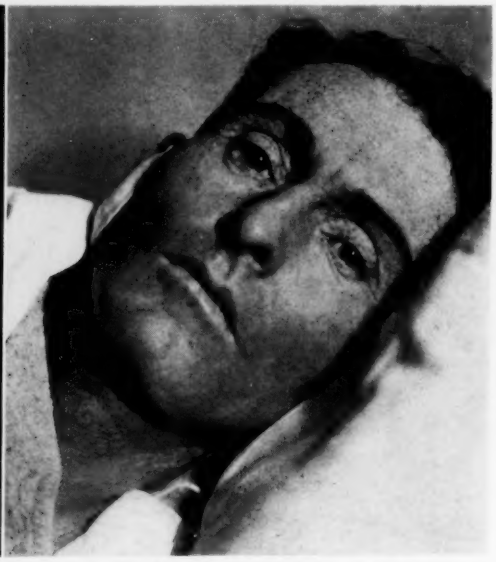

FIG. 4.—Photograph of patient shown in Fig. 3, on the day following section of the branches of the nerve to the orbicularis, frontalis and muscles of the upper lip. The annoying spasm is completely relieved.

mandibular branch of the nerve after section and immediate suture of the upper trunk which supplies the branches to the brow, orbicularis and upper lip. Preservation of the lower branch keeps the mouth balanced and prevents the unilateral smile which is so disfiguring (Figs. 3 and 4). The slight spasm which may persist in the depressor muscles of the mouth and the platysma is not troublesome and appears to diminish after the more severe contractions of the upper face have been relieved.

We prefer section with immediate suture of the nerve to produce paralysis rather than the injection of alcohol. It is important to save the mandibular

branch, if not too much involved, to minimize the deformity, and this can be accomplished more precisely by section of the nerve than by alcohol injection (Fig. 5).

The deformity resulting from paralysis of the two upper branches of the nerve for the relief of facial spasm is not so marked as that following division of a healthy nerve. The facial muscles in long standing spasm acquire contractures so that they do not show the relaxation and flabbiness so common in Bell's palsy. The deformity of facial paralysis varies con-

FIG. 5.—Photograph of patient four months following section and immediate suture of the branches of the nerve to the upper lip, eyelids and brow on the left side. The deformity is not marked, although it is evident in the wider palpebral cleft on the left. The mouth is well balanced. Operation July 3, 1936.

siderably in different individuals. In older patients the flabbiness of the tissues accentuates the deformity caused by the paralysis.

In one of the patients in our series, who had severe bilateral spasm, the branches to the eye and upper lip were divided on one side with preservation of the mandibular branch. On the other side the trunk of the nerve was injected with alcohol. Although some spasm persisted in the mandibular branch it was thought best to preserve it to enable the patient to drink fluids (Figs. 6, 7, 8, 9 and 10).

Prior to the operation this patient was tested with bilateral injection of novocain into the nerve trunks to determine his ability to drink after both

FIG. 7. FIG. 6. FIG. 8.

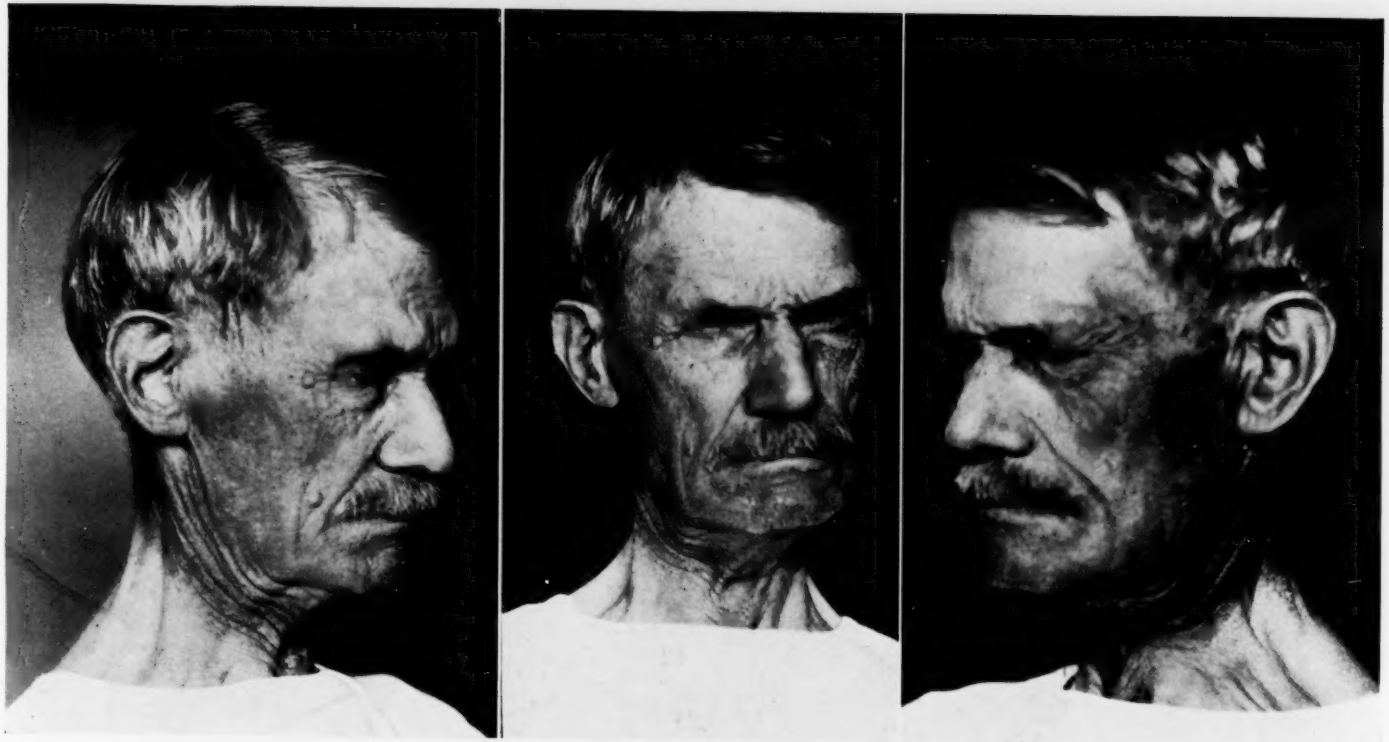

FIG. 6.—Male, age 74, with severe bilateral facial spasm involving the entire domain of both nerves. The muscles of the face were in almost continuous spasm with associated contraction of the platysma muscles. Contraction of these muscles depressed the chin, the upper lip was retracted and the eyes tightly closed during the spasm. The patient was unable to read and his walking was interfered with. Operation July 24, 1936. Temporary bilateral paralysis was produced by injection of 2 per cent novocain solution into the nerve trunk. It was found the patient had great difficulty in drinking. The nerve trunk on the left was injected with alcohol and a few days later the branches to the upper lip, eyelids and brow were divided under local anesthesia.

FIG. 7.—Photograph showing blepharospasm of the right side and contraction of the platysma.

FIG. 8.—Photograph showing left side of face with spasm of the orbicularis and platysma. None of the photographs was taken during a severe spasm.

nerves were paralyzed. This was the older of the two patients with bilateral facial spasm, a man 74 years of age. It was found, after the injection of novocain, that he had great difficulty in taking fluids. He may have acquired some expertness in this respect but he was a rather frail, undernourished man, and it was not thought wise to interfere too much with his ability to eat and drink. By preserving the mandibular branch he was inconvenienced very little in eating and drinking. (Figs. 6, 7, 8, 9 and 10.)

This patient was entirely relieved of the spasm for about six and one-half months following alcohol injection of the trunk on the left and section of the upper branch with immediate suture on the right. His general condition im-

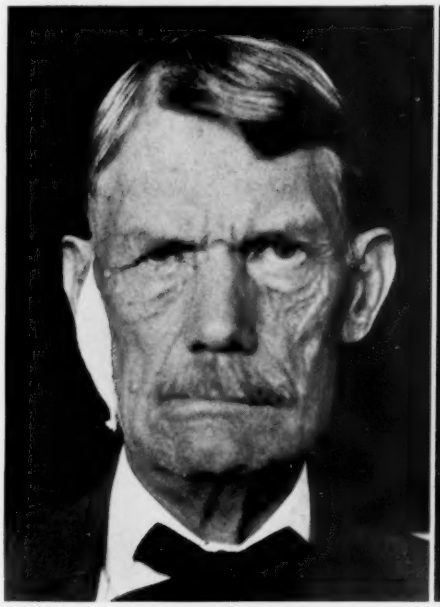

FIG. 9.—Photograph of patient five days after bilateral facial paralysis following injection of the nerve on the left with alcohol and section and immediate suture of the branches to the brow, eyelids and upper lip on the right. The slight spasm in the mandibular branch is shown on the right side. This branch was preserved so that the patient might have less difficulty in eating and drinking.

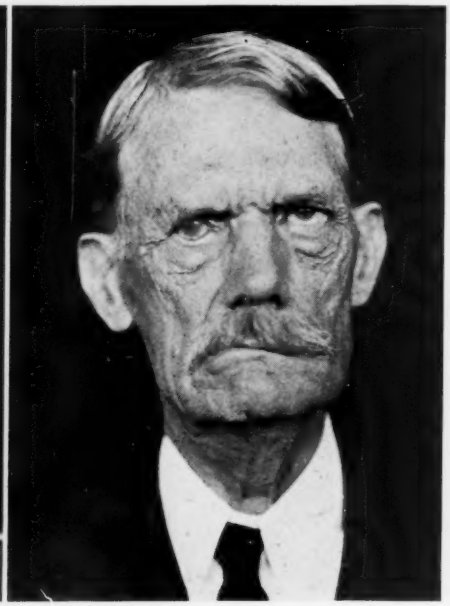

FIG. 10.—Same patient as shown in Figs. 6, 7, 8 and 9, three months after the production of bilateral facial paralysis with the exception of the platysma and the muscles of the right lower lip. There is evidence of slight spasm persisting in these muscles but this did not inconvenience the patient. No return of function in the paralyzed muscles. There is slight sagging of the lower lid on the right. Faciohypoglossal anastomosis as a primary procedure would restore function to the paralyzed muscles before atrophy had become advanced and is especially indicated in older patients.

proved greatly during the period of relief of the spasm. In February, 1937, the spasm returned and gradually became as severe as it was before the bilateral paralysis was produced in July, 1936. The paralysis following section and suture of the nerve on the right side lasted longer than that of the left facial nerve which was injected with alcohol. The spasm of the orbicularis oculi on the left was more disabling to the patient because the right eye had been practically blind for forty years, due to corneal ulceration.

The patient was readmitted on April 9, 1937. Operation: April 10, 1937.

Under local anesthesia a faciohypoglossal anastomosis was effected on the left side. The cervical sympathetic chain was divided just below the superior ganglion and immediately resutured. Three days after operation the patient was making a good recovery.

In the second patient with bilateral facial spasm, operated upon by my associate, Dr. W. G. Crutchfield, one side was injected with alcohol after exposure of the nerve. About two months later the nerve trunk on the opposite side was sectioned and immediately sutured. This patient did not experience much trouble in taking fluids, and, as is always the case, he did not have as much facial deformity from bilateral paralysis as is present when

FIG. 11.—Photograph of child, age nine, with congenital, bilateral facial paralysis. There is practically no facial deformity but characteristic absence of facial expression is noted.

paralysis is of the unilateral type (Fig. 11). When examined about 12 months following section of the nerve, there was evidence of some return of the spasm but the patient was not sufficiently annoyed by it to desire anastomosis.

Section with immediate suture of the nerve for facial spasm is somewhat analogous to alcohol injection of the trigeminal nerve for the temporary relief of tic douloureux. Recurrence of the disorder is almost inevitable in both conditions upon regeneration of the nerves. Permanent relief of facial spasm can be obtained only through changing the source of the innervation to the involved muscles by anastomosis of the facial with another motor cranial nerve.

For this anastomosis we have always preferred the hypoglossal (Figs. 12 A, B, and C) because of its functional similarity to the facial.[4] The results of faciohypoglossal anastomosis are very satisfactory in so far as recovery of motion on the affected side is concerned. There is evidence of improved tone in the face after about three months following anastomosis, and in about three and one-half months the first feeble contractions are seen about the angle of the mouth. Recovery of the muscle groups takes place from below upward with the function of the orbicularis returning last. We have never seen a case of faciohypoglossal anastomosis in which the frontalis muscle resumed function. Although faciohypoglossal fusion is uniformly successful in restoring motion to the paralyzed muscles, it does not restore emotional expression. A fair imitation of emotional expression may be acquired by reeducation but it lacks spontaneity and genuineness.

Kennedy,[5] in 1899, reported a case of facial spasm treated by facio-accessory anastomosis. Gibson,[2] in 1921, used the hypoglossal for the same condition. Wilfred Harris recommends faciohypoglossal fusion for the relief of facial spasm and Adson prefers the spinal accessory which he has used in two unreported cases. To narrow the lid cleft following section of the nerve, Leriche,[6] Sicard, Robineau and Haguenau,[7] and Adson[8] recommend simultaneous section of the homolateral cervical sympathetic chain. This produces a recession of the eye, a slight droop of the upper lid and lessens the lacrimal secretion which is sometimes annoying when the lower lid is paralyzed.

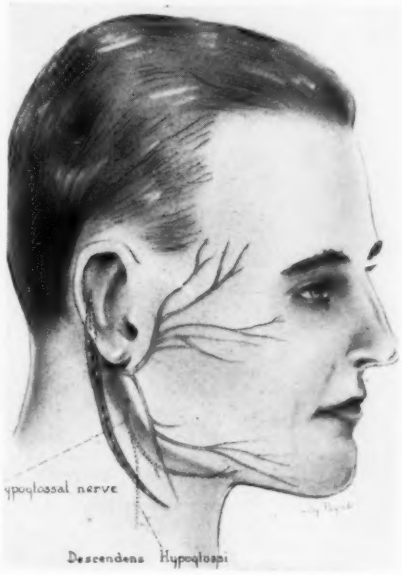

FIG. 12A.—Faciohypoglossal fusion. Position of incision for exposure of the facial and hypoglossal nerves. Incision begins just over the mastoid and curves downward and forward following, when possible, a skin crease.

In severe cases faciohypoglossal anastomosis should be employed as a primary procedure to effect a permanent cure of the spasm. If the condition is bilateral, the hypoglossal should be used on one side and the spinal accessory on the other. However, in some cases, sudden complete bilateral facial paralysis interferes with eating and drinking. To determine the amount of interference with these functions, brief paralysis of the entire face should be produced by injecting both facial trunks with 2 per cent novocain solution and then testing the patient for eating and drinking. If much difficulty is experienced by the patient, the operation should be performed in two stages, the second following about six months after the first.

Nerve anastomosis was recommended in two cases of the group under consideration. In one it was recommended after return of spasm following

section of the nerve and in another it was suggested as a primary procedure. All of the patients insisted on a preliminary section or an injection of the nerve with alcohol. The psychology of the situation is such that, after full explanation, I am willing to leave the choice of the procedure to the patient.

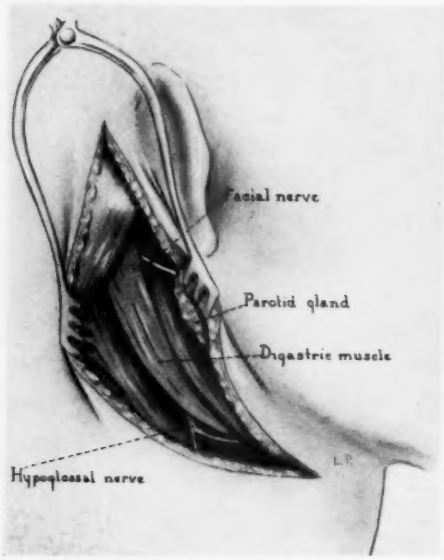

FIG. 12B.—Faciohypoglossal fusion. The sternomastoid muscle is retracted backward. When the mastoid tip is long it is sometimes removed with a chisel for better exposure of the digastric fossa. The hypoglossal is located just posterior to the cornu of the hyoid bone and traced backward but not divided until after the facial has been exposed and sectioned as near as possible to its exit from the facial canal. The best guide to the trunk of the facial is its small branch to the posterior belly of the digastric muscle. The facial trunk is seen as it leaves the digastric fossa. At the lower angle of the incision the descendens hypoglossi and main trunk of the hypoglossal are shown.

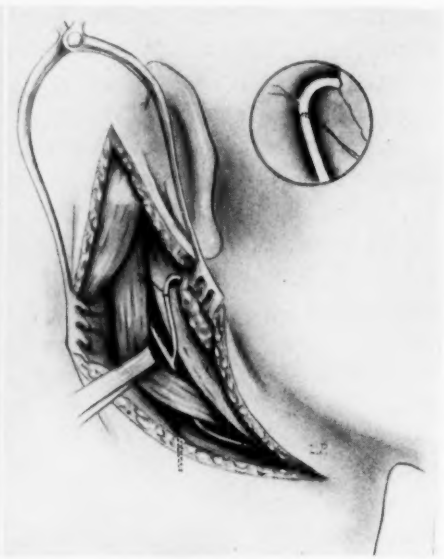

FIG. 12C.—Faciohypoglossal fusion. Anastomosis of the proximal end of the hypoglossal with the distal end of the facial is shown. Three or four silk sutures are sufficient to make accurate approximation of the two nerves. The drawing shows suture of the descendens hypoglossi to the distal segment of the hypoglossal. This is done to prevent atrophy of the tongue on the operated side. It is usually necessary to trace the hypoglossal back to the point where it winds around the occipital artery so that the segment of the nerve for anastomosis with the facial will be sufficiently long to unite the two nerves without tension. The trunk of the hypoglossal is divided just distal to the thyrohyoid branch but before section of the hypoglossal is made the facial trunk should be divided and placed in position for anastomosis. The anastomosis is covered with the digastric muscle.

CONCLUSIONS

The relief of facial spasm can be accomplished only by the infliction of the penalty of facial paralysis. The features of this paralysis should be carefully explained to the patient before the operative procedure is undertaken.

Although the effects of nerve anastomoses in the treatment of facial paralysis are well known, the final results of such anastomoses for the relief of facial spasm seem to have received little attention.

The paralysis necessary to the permanent cure of facial spasm should be relieved and this is possible by anastomosing the facial with the hypoglossal or spinal accessory nerve. This procedure offers the only chance of satisfactory relief of facial spasm.

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DIRECT ROENTGEN RADIATION OF BRAIN TUMORS DURING OPERATION

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SOME brain tumors—the meningiomata—are perfectly encapsulated and can be completely removed, and there are some types of gliomata that can be similarly treated. There are, however, a good many gliomata, fully 25 per cent, that are likely to recur even after they have been apparently completely removed. A very few of this type have been permanently cured. Some have had relief for several years but sooner or later they recur. How to cure these cases has given neurologic surgeons much concern.

In the hope of destroying any tumor cells that may have been left, roentgenologists have been using larger and larger doses of roentgen radiation. These have undoubtedly been effective in retarding a tumor's growth. One type of glioma—the medulloblastoma—is particularly susceptible to the roentgen ray, as was first pointed out by Sossman, working with Cushing, a number of years ago. Brain tumors, however, develop a resistance to this type of radiation and, though their growth may be retarded, up to the present time a cure has not been effected. Our practice has been to give as large an initial dose as possible in the hope that any cells that have been left may be destroyed. The obstacle to extremely large doses has always been the danger to the skin and bones of the skull.

Several years ago Drs. Sherwood Moore and Vilray Blair[1] began treating malignancies of the neck by administering the roentgen radiation directly into the open wound during the course of the operation. Doctors Moore and D. K. Rose[2] have done similarly in cases of carcinoma of the bladder. The results in both types of cases, malignancies of the neck and carcinoma of the bladder, were so encouraging that Doctor Moore has been urging me to try the method on cases of brain tumor. We knew that the normal brain tolerates a good deal of roentgen radiation and we had given as much as 600 R (measured in air) at one treatment per day, given to each of two to four areas.

In June, 1935, at a meeting of the American Neurological Association, in considering the results of the roentgen treatment of 119 gliomata,[3] I suggested that: "Larger doses of roentgen radiation should be used in the treatment of tumors of the brain," and stated that one method would be to devise "a safe method of giving therapy into an open cranial wound, thus avoiding the danger to the scalp and bone flap and delivering a much larger quantity of roentgen radiation into the tumor bed."

In 1936, a boy presented himself from whom we had removed a medullo-

FIG. 1.—Showing the manner in which the patient is draped, with heavy lead covering the bone and skin margins. This entire area is then covered with two sterile sheets, and the area to be rayed is marked on the sheet with a colored pencil.

FIG. 2.—Deep therapy being applied. The cone of the tube is placed directly over an area marked on the sheet, corresponding to the skull defect.

blastoma two years previously. Whenever his symptoms recurred, roentgen therapy checked them, so that we knew we had a very radiosensitive tumor. Finally we reached a point where his skin would tolerate no more radiation. This was our first opportunity to try the new method. His old wound was reopened May 25, 1936, and all vestiges of tumor were removed. Fortunately, there were no visible signs of subarachnoid implants which are so frequently found at a secondary operation for this type of tumor. With the wound open and all bone and skin heavily protected with four layers of lead, after suitable sterile towels had been placed over the wound, he was given 4,000 R. His wound healed without any reaction and he was discharged in less than three

FIG. 3.—A wound, two weeks after deep therapy had been applied, showing perfect healing.

weeks. We could discover no symptoms after operation indicating any interference with cerebral function so that we feel justified in concluding that this large dose had no ill effect on the normal brain tissue.

Since then, we have treated eight other cases of brain tumor similarly. One, a little boy, who also had a medulloblastoma, received 2,500 R; due to his restlessness we could not give more. Two other patients, who had a spongioblastoma multiforme, which is the type of tumor that gives us the greatest concern since it is not as radiosensitive and has a great tendency to recur, received, 4,000 and 5,600 R, respectively. Cases that have been operated upon some time ago we must treat in this way. We have realized that none of these cases was ideal for this type of treatment since they had all had a large amount of roentgen radiation to the tumor bed through the

skin and bone, and therefore the tumor might well have become radio-resistant.

The ideal way in which to apply this method is, of course, at the time of the initial operation and not at a second one. This is the method we are now employing whenever possible. We have had, to date, five cases, in each of which we have given 6,000 R, *without any filter,* directly into the wound during the course of operation. In four of these the K.V. used was 85 and in one 165. All these patients tolerated this huge dose very well and have shown no untoward after-effects. Their wounds have healed without any disturbance whatsoever. To prolong a patient's operation naturally was a matter of much concern. In our earlier cases the time of exposure lasted as long as one and a half hours. Doctor Moore has, however, by using *no filters whatever* cut the time down so that he is able to give 6,000 R in from 30 to 40 minutes.

We make no claims for this new method as yet, but merely wish to present it. If roentgen radiation is going to help in stopping deep seated, malignant growths of the brain, huge doses must be used, and this method makes it possible to give doses far in excess of anything that has previously been possible. We want to reemphasize the fact that these large doses have apparently had no harmful effects on the patient.

We hope to make further reports on this method after sufficient time has elapsed to determine end-results.

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THE TREATMENT OF BRAIN TRAUMA

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Swift[1] states that there are approximately 112,000 cases of skull fracture occurring annually in the United States with an average mortality of 25 per cent. Wortes and Foster Kennedy report a mortality of 37.8 in 1,000 cases of cranial injuries admitted to the Bellevue Hospital between 1920–1925. Fred Besley, of Chicago, in a report published in 1916 by the Cooke County Hospital, regarding 1,000 consecutive patients having fracture of the skull, states that the mortality was 53 per cent. When we remember that during the Great War the United States had 36,694 killed and that 13,691 died of wounds, or a total of only 50,385 deaths in battle, we must realize what a serious calamity to our county the mounting death rate from accident is. Many statisticians believe that approximately 125,000 injuries involving the cranium and its contents occur in the United States annually.

My object in presenting this subject is not so much to attempt to bring out something new in the treatment of brain trauma but rather to give a brief résumé detailing the treatment of such cases over the period of the past 25 years, hoping that it may aid in giving information and encouragement to many doctors throughout the United States who are called upon, only occasionally, to treat and manage acute traumatic brain lesions.

Primarily, these patients should be treated where they receive the injury, if possible, because to move them roughly might cause death. Immediate treatment by the local doctor for the shock is often life saving. We have had brain trauma cases brought to our clinic in automobiles, with death resulting soon after admission, the patients not having been critically injured, apparently, but they could not, however, survive a 25 mile auto trip.

The neurosurgeon treats a very small percentage of these cases of traumatic brain lesion. If he could, the mortality and morbidity would unquestionably be markedly reduced. We are greatly indebted to these specialists for their teachings and for the industrious research work that they have accomplished. It is to be regretted that the profession generally is not better informed regarding their efforts, although I wish that they themselves might agree more fully as to the best method of treatment for these cases.

Within a radius of 120 miles of Chattanooga, there is a population of approximately a half million, and there is not a neurosurgeon living within this area; which shows the necessity of a better understanding by the general profession of how these cases should be managed. Doubtless there are many other such areas in the United States which are devoid of these specially trained men.

In 1922, I[2] reported that: "We have treated in our hospital during the past five years 68 cases of serious brain injuries that we have tabulated. We

have not included all the brain injuries that we have received into the hospital, as some were moribund when brought in and died within a few hours, and some which were never serious were not included, but we have included all the brain injuries that were operative; some of these were deeply unconscious and greviously ill at the time of operation. The nonoperative brain injuries were usually the ones with linear fractures, or no fracture, intracranial pressure not high, either not unconscious or only moderately so, and no focal symptoms of localized hemorrhage, and no depressed fractures—the class of case that without complication and without other injuries should recover.

"There were 45 operative cases of brain injury—subtemporal decompressions—with 14 deaths (31 per cent mortality). These were the most serious cases of the series.

"There were 19 cases not operated upon, with five deaths (26 per cent mortality): three of these deaths occurred suddenly and were unexpected; two were treated by repeated spinal punctures to relieve the increased intracranial pressure—both recovered."

It is possible that the three unexpected deaths were due to the late effects of spinal puncture, as in this era of our work we performed spinal taps in all cases where we suspected a marked increase of intracranial pressure.

The headaches, restlessness and mild unconsciousness resulting from intracranial pressure when not relieved by dehydrating measures, are often relieved by drawing off from ten to 25 cc. of spinal fluid, under the control of a manometer, not allowing the pressure to go below ten to 15, which has, occasionally, been spectacular in its favorable results. As a result of the teachings of many neurosurgeons, we have the utmost respect for spinal decompression by withdrawing the spinal fluid in cases of traumatic brain injuries, and it is most probably due to this respect that we have not had any deaths, to our knowledge, following this procedure.

In order to determine the results of our present method of treatment as compared to those obtained over a 15 year period earlier, an analysis was made of the last 100 consecutive cases coming under our care. In this series were included those which survived two hours or longer after admission to the clinic. In 56 per cent of the entire series the injury was due directly to automobile accidents. The next most frequent cause were the results of industrial accidents (25 per cent). Fights, during which blows on the head were administered by various weapons, were responsible in 12 per cent, and falls, causing cranial trauma, accounted for the remaining eight per cent.

It has long been our contention that the way these cases are handled by laymen immediately following the accident has a most important bearing on their condition. All of us are familiar with the totally unconscious patient, who on recovering from the initial shock, lying quietly and breathing normally, is moved roughly, or has his head raised quickly; the patient begins vomiting and straining immediately, respiration becomes rapid, and it is several minutes before they recover. In 88 per cent of this series, trained ambulance attendants brought the patients to the hospital, and we feel that

the subsequent recovery of many was due to their proper and careful transportation.

Our first concern with the brain trauma case is the determination of the presence or absence of shock. The grave possibilities of this feature are, far too frequently, not appreciated. Shock was present in 36 per cent of this series. No examination or manipulation of any kind was attempted until definite recovery from this condition. The usual measures are employed, with stress placed on absolute quiet, warmth, one-fourth grain morphine hypodermatically and intravenous injection of 50 to 300 cc. of from 10 to 50 per cent glucose.

We do not attempt, at first, to classify our cases according to the type of pathology present, but try to consider the clinical signs and symptoms as a whole in regard to the method of treatment indicated.

In 75 per cent of this series the clinical syndrome present was that described by most authors as concussion. It is not within the province of this paper to discuss the many and varied theories as to the actual pathology present in this condition. We are content to use the word concussion by preference, as the laity and the legal profession know what we mean by concussion. This clinical syndrome is usually characterized by very temporary unconsciousness, or an unconsciousness lasting several hours, followed in some cases by vomiting, severe headache, and vertigo. Recovery is prompt and complete under proper management. Absolute quiet, with rest in bed, is essential to allow the proper readjustment of cellular and fluid content of the brain. When possible, these patients are kept in bed ten days. We feel that this usually shortens the period of disability because recovery from headache and dizziness is far more prompt. It is our practice to give these cases magnesium sulphate by mouth on the second or third days. We use codeine and luminal in small amounts for pain and restlessness. A light diet is allowed during hospitalization.

In the more severe type of brain injury, the symptoms and signs are variable. It is imperative for the attending physician to be able to correctly correlate his findings, in order to properly undertake treatment. In this type of injury, many authors attempt to classify each case separately, as laceration, contusion, edema or hemorrhage of the brain. Any severe force applied to any portion of the cranium is transmitted with very little loss through the entire quantity of the contained fluid. So that trauma over a lateral ventricle may very well cause multiple petechial hemorrhages in the fourth ventricle, or in the cisterna magna or cisterna basalis. Obviously, then, most cases of severe brain trauma are a combination of two or more of the previous named types, namely, laceration, contusion, edema, or hemorrhage.

Of the 100 cases, 25 were considered as severe brain trauma cases, and of this 25, 20 were considered as severe combined injury to the brain. Following recovery from shock the patients were carefully examined. There are a number of signs and symptoms which must be evaluated in order to determine the best method of management. To us, the most valuable of these have been the

following: (1) The stage of consciousness; (2) stage of restlessness; (3) the pulse; (4) the temperature; (5) the respiration; (6) the blood pressure; (7) the presence or absence of paralysis; (8) pupillary changes; (9) bleeding, and (10) multiple injuries.

An accurate notation of each of the above items, at the first examination of a brain injured case, affords a most excellent method of observing what changes occur in the patient's condition during the hours, or days that follow. All of these 20 cases were unconscious when brought to the hospital. A change in the stage of consciousness is probably the most important single item indicating the patient's progress. Restlessness, present in 55 per cent, is always a bad sign; to us, it indicates a general cerebral irritation either from a marked edema, diffuse bleeding, or extensive contusion. The rate and quality of the pulse should be carefully noted. A very slow pulse, as a rule, indicates an increase in the intracranial pressure. Following recovery from shock, it is our experience that a very rapid pulse is a grave prognostic sign. High temperature following shock is usually an indication of impending disaster. Rapid respiration is also a bad prognostic sign. A record is kept of blood pressure changes, but, to us, has not afforded as accurate an index to cerebrospinal fluid pressure as some authors claim. The presence of pupillary changes or paralysis usually indicates a localized lesion and will be discussed later under operative indications. Bleeding from the nostrils or ears was present in ten instances. This, at times, indicates basal fracture and severe brain damage. Multiple injuries complicated brain trauma in 44 per cent of the 100 cases. Great care must be used in treating these injuries in the stage of serious cerebral embarrassment. It is much better to delay treatment until partial recovery has occurred.

As a diagnostic aid, the roentgenologic examination is of prime importance where a depressed fracture is suspected. In cases where there is no depression we do not feel that a roentgenogram of a fracture of the skull is of great value, but it is of importance in diagnosing brain injury, as certainly a fracture running through the middle fossa is of far more importance than one involving the anterior fossa. However, we do not insist on roentgenograms being made until the patient is well able to withstand the procedure. Most of our examinations are taken by a bedside unit, with the patient in bed, and it is our practice to make them where no real injury can result to the patient; not only because of their diagnostic value, but also to complete the record, and for medicolegal purposes.

With us it is frequently difficult to convince the patient that he is seriously injured and should remain in the hospital under constant observation, unless we can definitely assure him that the roentgenogram shows he has a fractured skull.

The severe brain injuries in this group were all treated conservatively. They were put to bed immediately and absolute quiet demanded. Intelligent and well trained nurses were kept in constant attendance. It is rare that we

put these restless cases in a strait-jacket as we have found that the restriction irritates the patient and increases his irrationalness and uncontrollability.

The value of strong solutions of intravenous glucose is questioned by many competent authors. However, we feel that the physiologic reasons for the use of these solutions are well founded. Solutions of glucose ranging from 25 to 50 per cent in strength and usually from 50 to 100 cc. in amount are given to our patients every four to six hours. We feel that these injections are of definite value, and there have been no recorded ill effects from their use. Sedation was satisfactorily accomplished, in most cases, by the use of sodium luminal in five grain doses, either intramuscularly or intravenously. No concern is felt in giving large doses when necessary. Morphine, after the initial injection for shock, is not used because it is feared that it masks any symptoms that might develop and further depress an already embarrassed respiratory center. Small pledgets of sterile cotton saturated with alcohol are placed in the external auditory meatus when there is bloody discharge. Bleeding from the nostrils usually stops quickly; however, on two occasions it was necessary to pack the nostrils to control it. The packing is effected with vaselinized gauze as it causes so much less trauma in removing than does dry gauze. This is a dangerous procedure as it is likely to lead to infection and resulting meningitis.

While spinal puncture is a very important diagnostic and therapeutic aid, we do not feel that it is indicated routinely. Our experience indicates that the indiscriminate withdrawal of cerebrospinal fluid does far more harm than good. When the patient exhibits extreme restlessness, marked slowing of the pulse, rapid respirations, or increasing unconsciousness, indicating advancing edema or diffuse hemorrhage, we employ spinal puncture for both diagnosis and treatment. This, however, was done in only five cases in this series. On one occasion, repeated punctures were considered necessary, each time bloody fluid was obtained, and it was felt that the withdrawal of this blood hastened recovery.

Liquids in moderation are administered to the patients by mouth as soon as they will swallow them when placed on the tongue. Magnesium sulphate in two to three ounces of a 50 per cent solution is given by mouth or by rectum on the second or third day, and is repeated frequently to aid in dehydration.

These patients have frequent daily observations made while they are hospitalized. Further treatment depends on the daily change in signs and symptoms. Usually these cases will need catheterization for the first 24 or 48 hours, following which they void involuntarily at irregular intervals. It was interesting to us to note that in the 25 severe cases the average time of unconsciousness was 5.7 days and the average period of involuntary micturation was 5.2 days. When these patients void voluntarily we feel much easier about their condition. All severe head injuries are kept in the hospital until they are fairly well conscious and oriented. The average period of hospitalization in this group was 17 days.

A small percentage of head injuries offers different indications for operative interference. The most outstanding of these, of course, is decompression. This type of fracture may be accompanied by numerous complications, such as: Compound injury of the scalp, severe comminution of the bone, or direct laceration of the brain and meninges.

It is our usual method, in recent depressed, compound injuries, to débride thoroughly, remove all small unattached bone fragments, wiping away contused brain tissue with moistened cotton pledgets, control bleeding from the brain with fine silk ligatures or silver clips, close the dura with fine silk and repair the scalp in layers without drainage. In probably infected cases, we simply cleanse and débride the wound carefully and thoroughly, repair the dura and pack the scalp edges lightly with vaselinized gauze. A secondary closure is effected later if no infection develops. Should there be no scalp injury present and if the patient presents no symptoms of pressure, we wait several days before elevating the depression. All of these operations are performed under 1 per cent novocain anesthesia.

The other indication for operative procedure is localized severe extradural hemorrhage, usually encountered following injury to the middle meningeal artery. The syndrome described in text-books is usually easily recognizable but unfortunately is seldom present. Without deviations, the lucid interval after injury followed by unconsciousness, dilatation of the ipsilateral pupillary paralysis of the contralateral extremities, decreasing pulse rate, *etc.*, may all be present in these cases and do constitute operative indications. However, it has been our experience that it is usually a diffuse injury to the brain which accompanies the above picture, and complications should be guarded against in most instances. In several cases in which I have operated, a tense dura was found, and almost clear cerebrospinal fluid, under great pressure, spouted up when the dura was incised. Such cases are usually quickly relieved, as a rule, simply by relieving the pressure.

Of the group of 25 severely injured patients, six came to operation. Four of the five depressed fractures were elevated with excellent results. The fifth was a very slight depression in the temporal region and operation was not judged necessary. In two cases a diagnosis was made of middle meningeal hemorrhage and exposure of the involved area was effected under local anesthesia. In each instance, extensive laceration and contusion of the brain substance were found and both cases succumbed a few hours later.

Of the 100 cases in this series, there were five deaths, a mortality of 5 per cent, and in the severely injured cases, 20 per cent. All of these cases died within 96 hours following injury. The two cases on whom decompression was performed for extradural hemorrhage had evidence of severe contusion and laceration of the brain and in two of the remaining three cases we did not feel that any type of treatment would have helped. One of the cases included in the mortality list was very interesting and instructive to us. A young, robust man received a moderate blow on the left parietal region, with the small end of a billiard cue. The outer layers of the scalp were split for

approximately one and one-half inches. The patient walked five blocks to the emergency room, the laceration was dressed and he returned to town where he remained four hours before walking one and one-half miles home, where he went to bed. The following morning, at 7 A.M., a reporter was calling the clinic asking rather embarrassing questions about who treated the man who was found dead in bed at 6:30 A.M.

Of the 20 patients surviving severe head injury, accurate follow up records have been obtained in 16. Eleven of these write that they are entirely free of symptoms in any way pertaining to their injury; three of the patients complain of headaches, vertigo, and weakness. Without exception these symptoms occur following exposure to heat, excitement, or undue exertion. Small doses of luminal are very valuable in such instances. In one case there was a permanent paralysis of both external recti muscles. One patient who had a severe injury to the scalp and brain has definite character and personality changes, with convulsions following exposure or excitement. This incidence of sequelae corresponds very well to that reported in the literature by other authors. Patients with definite convulsions, at times, require the services of a skilled neurosurgeon, but, as a rule, very little can be done to alleviate their complaints.

Alton Ochsner[3] states that: "In the Charity Hospital series, which we studied, very few operations were performed and the results obtained in this group demonstrate the value of conservative treatment. In the decade from 1910–1919, only 5.2 per cent of the cases were operated upon, the incidence of operation decreased in the next decade to 1.6 per cent, and in the two years, 1930–1931, the incidence of operation was 0.5 per cent. In the entire series of 1,099 cases only 31 operations were performed, an incidence of 2.8 per cent. The value of this ultraconservative treatment is shown by the results obtained in our series. In the entire group of 1,099 cases there were only 92 deaths, 8.4 per cent. If one excludes those cases dying within the first 24 hours, in which probably relatively little could be done, because of the extensive injury, there were only 43 deaths, a mortality rate of 3.9 per cent. The mortality rate in the operative cases was 25.8 per cent, whereas, that in the nonoperative cases was 7.8 per cent."

I do not believe that the 53 per cent mortality rate reported by Besley from Cook County Hospital, and the 3.9 per cent mortality rate reported by Ochsner represent the difference in treatment, but rather represent the statistician's compilation. I do not believe that cases of concussion without complications should be included in any series representing the treatment and mortality of head injuries. Neither do I think that a case of skull fracture without hemorrhage or marked increase of intracranial pressure should be included in any such series, as practically no active treatment is indicated in either case. To properly evaluate the difference in mortality series under the various methods, only severe brain trauma cases should be included. In our series, reported in 1922, treated principally by subtemporal decompressions and spinal taps, our mortality rate was 31 per cent. In analyzing cases

reported 14 years later and treated by conservative methods, the mortality rate was 20 per cent. Both series were treated by the author personally and it is fair to say that the difference in treatment has reduced the mortality 11 per cent.

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DISCUSSION OF THE PAPERS OF DOCTORS COLEMAN, SACHS, AND NEWELL

DR. JOHN T. MOORE (Houston, Tex.).—I have enjoyed all three of these papers, but I wish to make a remark about Doctor Sachs' paper regarding the direct application of roentgen therapy to these tumors and to suggest—I would like to see it tried—that the surgeons make more use of interstitial short wave roentgen ray and radium in the treatment of all malignancies. Using one and two milligram platinum needles properly placed, as a part of the operation, leaving them there two, three or five days, as you can safely do, would accomplish a great deal where radiation is indicated. I would like to have Doctor Sachs consider the use of these platinum covered needles at the site of his tumor as giving the most effective radiation. I am glad to see that we are using, more and more, the direct exposure of tissues to roentgen ray and radium radiation.

DR. KENNETH H. AYNESWORTH (Waco, Tex.).—I live in one of the districts Doctor Newell described, in the center of an agricultural district with a population of 250,000. In that district there are about 20 hospitals and four of these are in Waco. We have an enormous number of injuries to the skull and brain brought into these hospitals. That means that these patients receive the treatment that the ordinary surgeon would give.

At a meeting of our District Medical Society a year ago, a paper was read by one of our surgeons who suggested such radical measures that some of us older men thought we should combat them. He advocated taking each patient to the operating room immediately upon admission to the hospital and operating. I said there was not a neurologic surgeon in our district, and these patients should be put to bed and kept there as quiet as possible and that nothing should be done unless there was visible hemorrhage, which should be stopped by the easiest possible method and the wound, if there was one, treated; that the patient should be treated conservatively until a proper consultant could be called. By conservatively I mean without the use of morphine if possible. Since that time I have noticed a marked diminution in the death rate in these cases.

I had seen what I thought were bad results due to the moving, immediate roentgenographic examination and operation in these cases. Except in cases of serious hemorrhage, it occurred to me that operation was the wrong thing in the hands of the type of men who would have to perform it. Since we have adopted the conservative method of handling these patients, the mortality has dropped markedly. This gives us time to call in someone better qualified to handle the more serious cases, if and when necessary.

DR. ERNEST SACHS (St. Louis, Mo.).—It is very gratifying to see the general surgeons take such a lively interest in the treatment of head injuries,

for we have had that on our minds for many years. I think all surgeons are faced with the question of what to do. It is true, perhaps, that in certain details neurosurgeons differ somewhat, but I think we agree on the general principles. What was just said I think is very pertinent: First, not to be in a hurry about making roentgenologic examination of these cases. Second, to keep them quiet and not be in a hurry about treatment, especially about surgical procedures. In the majority of cases I think surgery is not indicated. I differ with some of my colleagues in that I believe the thing to consider is the treatment of the edema. You cannot do anything for a lacerated brain, but you can do something for cerebral edema. Those who have criticized the use of hypertonic solutions, either magnesium sulphate by rectum or the use of sucrose, I think are in error, for when given repeatedly this method undoubtedly reduces the cerebral edema. If the treatment is only used once the edema will recur and it must be repeated.

Dr. R. Glen Spurling (Louisville, Ky.).—So much might be said about head injuries that it is difficult to know where to begin and where to leave off. Doctor Newell's paper has summarized the subject clearly, concisely, and I think conservatively.

Attempts to lay down rules for the management of every case seem to me futile. I know of no other condition in medicine where a nicely weighed judgment is more important. I think too much has been said about conservative and radical treatment. As a matter of fact, the so called conservative measures may be extremely radical if improperly applied. The chief controversy seems to center around spinal punctures, operations and dehydration. In my experience, each one of these methods when thoughtfully applied is an important therapeutic agent. When done routinely they may be not only useless efforts but positively harmful. Careful neurologic observations will usually indicate those patients in whom these forms of treatment are indicated.

The term "edema of the brain" has been used entirely too loosely in the discussion of head injuries. Unfortunately, with the wave of enthusiasm over conservative treatment, many patients have been subjected to dehydration by every conceivable means early in their illness. I doubt seriously whether edema of the brain is ever early in onset. In an attempt to get some information on this subject during the past year we have taken routine spinal fluid pressures on all head injuries that entered the Louisville City Hospital. During the first 24 to 48 hours, high spinal fluid pressures are seldom recorded except in those patients who have a surgical complication (extra- or subdural clots, subdural hygroma or intracerebral clots). Most severe head injuries have a low or low normal cerebrospinal fluid pressure. To give these patients hypertonic solutions intravenously is to me definitely bad therapeutics. We keep the fluid intake at a moderately high level in such cases during the period in which the spinal fluid pressure is low. After 48 hours some patients do have increased intracranial pressure either from edema of the brain or from meningeal irritation from blood. In such circumstances dehydration is a logical and important phase of treatment. Of course, hypertonic dextrose should have been completely supplanted by hypertonic sucrose. Hypertonic dextrose is invariably followed by a secondary wave of edema from concentration of the dextrose in the tissues of the brain. With hypertonic sucrose this secondary wave is eliminated because the sucrose is not metabolized and is excreted in the urine as sucrose.

Dr. Joseph E. J. King (New York, N. Y.).—I have enjoyed listening to Doctor Coleman's paper. It is the first good paper we have heard on this

subject. This most deforming, terrifying condition in an otherwise presentable individual who suddenly makes grimaces and monkey faces is very disheartening. Although I know nothing about the pathology, I think Doctor Coleman is right when he states that the pathology lies within the nerve itself and that it is not cerebral. Anastamosis of the involved branches of the facial with the hypoglossal seems to be more sensible than sectioning and resuturing the facial nerve.

I have had the good fortune to be acquainted with the late Doctor Duel and Doctor Tickle, who have done so much work on facial paralysis. They have obtained splendid results with transplantation. Doctor Tickle told me that for about six months or more everything looks all right, but after a while a number of the patients develop facial spasm. These patients had never had anything the matter with their brains, but the nerve had been injured at the time of a mastoid operation. Nevertheless, spasm followed transplantation of a section of nerve into the defect.

Doctor Sachs' use of radiation in cases of brain tumors directly into the open operative wound is certainly to be commended, and we shall most surely follow his suggestion. The danger of burning the skin has prevented proper radiation. It is common sense to believe that when a tumor has been removed, or partially removed, by morcellation, tumor cells are still in the neighborhood. It is inconceivable that all the cells can be removed, therefore it seems that the proper time to use roentgenotherapy is at the time of operation, and directly into the wound. We have never done this, but will attempt to do so hereafter.

I should like to say one word possibly in extenuation of the statistics presented by Doctors Kennedy and Wortis in the report which they made of craniocerebral injuries in Bellevue Hospital. This group included all types of craniocerebral injury regardless of their condition when they arrived, unless they were dead on admission. The group included all types of severe injury, and the cases were treated by a great number of men on the various divisions. The number of admissions is so enormous it is impossible for the neurosurgeons to handle them, so that they have been treated on the general surgical services. Recently these cases came under the control of Dr. Arthur Wright of the Third Surgical Division, and they are handled more specifically by Doctor McLean. I feel sure that the statistics to be reported hereafter will show improvement. Although a mortality rate of 35 or 37 per cent is rather high, I doubt seriously if, in a general hospital, one will be able routinely to obtain a mortality rate as low as 5 per cent.

Dr. James Barrett Brown (St. Louis, Mo.).—The patient Doctor Sachs referred to with carcinoma of the tongue was treated about 13 years ago by radical operation by Doctor Blair and by roentgen radiation through the open wound by Dr. Sherwood Moore. The patient lived seven years, but it is probable that the radiation had little to do with the cure of the disease. A few years ago, after many discouraging results in treatment of advanced malignancies of the neck, I asked Doctor Moore if he would consider treating these patients if the overlying skin was gotten out of the way for him. We did not have any very good results, but, now, after further observations, Doctor Moore thinks the plan might be more successful. Dr. D. K. Rose and Doctor Moore adopted the method of open radiation for tumors of the bladder and obtained some good results. The difference is, that here a primary growth is treated instead of a metastatic one as we started to do in the neck. The same thing applies in Doctor Sachs' work, where he is treating primary metastatic tumors in the brain, and some excellent results may be expected. Last week, in a patient, whom I operated upon for Doctor Blair, with an infiltrating basal

cell growth of the forehead and frontal sinus, I removed the remaining fragments of the frontal bone, and Doctor Moore gave 8,000 R at one time—directly over the frontal region—the patient left the hospital in good condition without any known cerebral symptoms resulting from the treatment.

DR. CLAUDE C. COLEMAN (closing).—I have been asked about the success of faciohypoglossal anastomosis. This anastomosis is uniformly successful in restoring motion to the paralyzed muscles, provided it is performed before these muscles are completely atrophied. The restoration of facial expression is limited after this operation but its success in this respect depends to a great extent upon the patient's efforts at reeducation. The facial nerve has a high capacity for regeneration.

In my experience, faciohypoglossal anastomosis, a few years ago, was nearly always required because of paralysis due to mastoid infection or following mastoid operation in which the nerve was traumatized. At the present time faciohypoglossal anastomosis is more frequently required because of an intracranial destruction of the nerve due to complete removal of cerebellopontine angle tumors. It is rarely possible to save the nerve when the capsule of these tumors is completely removed, although in a recent case a large tumor was removed with preservation of the nerve and restoration of its function after a temporary paralysis.

I have been very much interested in a nerve graft operation of Ballance and Duel for the relief of facial paralysis and I want to thank Doctor King, both for the remarks about my paper and for his reference to the Ballance-Duel operation. Nerve graft operation is not applicable in the treatment of facial spasm or in paralysis following removal of an angle tumor, but good results have been reported from nerve graft when the nerve is injured in the facial canal.

A careful explanation should be made of the patient with facial spasm before any operative procedure is begun, whether for simple section of the nerve, injection of alcohol or faciohypoglossal anastomosis. The details of facial paralysis should be carefully explained, giving due attention to the effects on the eye and other features of the penalty which the patient must pay for the relief of the spasm.

I want to emphasize that in view of the fact that paralysis is necessary for the cure of facial spasm, this paralysis should be treated precisely as that following removal of angle tumors, that is, by faciohypoglossal or facio-accessory anastomosis. Changing the innervation by substituting one of these cranial motor nerves for the proximal portion of the facial should relieve the paralysis without recurrence of the spasm.

DR. ERNEST SACHS (closing).—Regarding Doctor Moore's question about the use of radium, for a year and a half before we began using roentgen radiation we tried planting radium emanation all around, implanting as many as 25 seeds in the wall from which the tumor had been removed, and we have only one case that is still symptom free over two years. We found the method so unsatisfactory that we were glad to make use of the opportunity which Doctor Moore of St. Louis gave us of trying this other method, which I hope will be more successful and effective.

ANEURYSM IN THE CERVICAL PORTION OF THE INTERNAL CAROTID ARTERY

AN ANALYTICAL STUDY OF THE CASES RECORDED IN THE LITERATURE BETWEEN AUGUST 1, 1925, AND JULY 31, 1936

REPORT OF TWO NEW CASES

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SINCE extracranial aneurysm of the internal carotid artery is a notoriously treacherous lesion and especially since no surgeon has had a wide experience with the condition, the completion of its history together with an account of two hitherto unpublished cases may not be amiss at this time.

The first case of aneurysm in this part of the carotid system was observed by Sir Astley Cooper,[14] in 1808. For a long time, further observations filtered into the literature very slowly, but of late the occurrence of aneurysm in this situation has been noticeably on the increase and the affection has ceased to be a novelty. Thus, as recently as 1906, Bobbio[5] could muster only 18 cases, whereas, exactly 20 years later, one of us (N. W.)[68] was able to compile a series of 106 cases. In the meanwhile, counting the two cases herewith presented, 18 additional observations have found their way into current medical periodicals. Altogether this makes a total of 124 examples of extracranial aneurysms of the internal carotid artery on file. The condition is, therefore, occurring in sufficient numbers to demand the utmost caution in dealing with affections of the throat.

In speaking of arteriovenous aneurysm between the internal carotid artery and its satellite vein, von Bergmann[4] states that it is a rare lesion and cites only two cases, those of Giraldès[25] and of Joret.[38] Callander[8] in a more recent communication adds 10 additional cases. However, two of these cases (Chartier,[11] and Quénu's[58]) are fistulae between the primitive carotid and the internal jugular vein and do not belong in the group under investigation. Winslow[68] cites 19 observations in his paper. Of these cases only three were recorded prior to 1900.

We attribute the increased incidence of aneurysms in this part of the carotid system to the introduction of the high-velocity, steel-jacketed bullet which cuts its way through vessels and does not push them aside as is the case with the slowly moving lead ball.

The chief interest in the condition, however, does not reside in its comparative rarity. Of greater import to the clinician is the possibility of its being mistaken for a tonsillar abscess from its penchant for appearing in the

neighborhood of the tonsil, with little or no manifestation of its presence in the neck. As a consequence of this peculiarity, the unwary physician has lanced an aneurysm of the internal carotid artery, on occasion, for abscess, with the consequent death of the patient from hemorrhage.

Blunders of this sort are historic. Chassaignac,[12] Liston,[41] Vas,[64] Lee,[39] and Schlagenhaufer[59] have made this mistake. The patients of Minich,[47] Wulff,[69] Reid and of Winslow had similar experiences, but others than the authors committed the error. In the case reported by Minich, a bewildered physician lured by the appearance of the swelling incised it for an abscess and to his amazement was greeted with a gush of blood instead of pus. The bleeding was arrested with difficulty, but two days later a fatal hemorrhage occurred. Soon after the apparent recovery of Wulff's patient from an attack of angina, a physician noticed a tonsillar bulging which he cut for an abscess, but only blood escaped. The tumor soon returned and pulsated. Wulff, suspecting it to be an aneurysm, inserted a needle into the swelling and aspirated pure blood. Ligation of the primitive carotid cured the condition. Winslow's patient had a mass in the right side of the throat. Her doctor thinking it to be an abscess, lanced the swelling and evacuated blood but no pus. Much to the woman's chagrin, the lump continued to increase in size. Ligation of the right common and external carotid arteries effected a cure.

Wagner[67] was spared the humiliation of cutting into a swelling of the same character by neglecting to take along with him his instruments. When he returned, he learned much to his surprise that the patient had died of a hemorrhage into the mouth. Duke[19] barely avoided a similar disaster. He was about to open a supposed abscess in a patient's mouth when he detected an aneurysmal pulsation in the tumor. A consultant did not consider the condition so serious and lanced the swelling. There was a gush of blood, the arrest of which necessitated ligation of the primitive carotid. The patient died later of a secondary hemorrhage. Zamboni[70] was saved from a like catastrophe by his patient's refusing to be operated upon. Johnson[35] nearly committed the same blunder when he attempted to lance the swelling in his case, but had to desist on account of the child's unruliness. An associate of Helman's[29] intended to open such an aneurysm for an abscess. He sent the woman to the clinic for demonstration but advised her to return to have the swelling opened. According to Helman, the condition at first glance might have been mistaken for a tonsillar abscess, but its long duration, absence of pain and lack of inflammatory signs were against such a diagnosis; besides, the swelling was actively pulsating in unison with the radial pulse. A colleague suggested that a trocar be inserted to verify the diagnosis, a procedure which Helman deemed neither expedient nor safe.

An accident no less serious than meddlesome surgery is spontaneous rupture of the aneurysmal sac into the mouth. This catastrophe follows failure to recognize the condition or to ignorance concerning its proper

treatment. In the case reported by Pircher,[54] an external swelling was diagnosed as a cervical abscess. The patient's jaws were fixed and she could not open her mouth. Pircher made an incision in the neck but failed to reach the suspected pus. He thereupon called in consultants who agreed with him that the lesion was an abscess and advocated the insertion of a trocar. This procedure also failed to yield the desired result. The child died suddenly of a copious hemorrhage into her mouth. The symptoms were so obscure and misleading that Pircher completely failed to suspect the real nature of the affection, and he believed, until the death of the patient, that he was dealing with an abscess. At no time did he seem to sense what the trouble was and he was unable to account for the origin of the bleeding. It was only at autopsy that he became aware of the character of the lesion. Perier[52] considered the condition so perplexing that he was unable to decide upon the true character of the malady in a case under his care. The patient succumbed to a profuse hemorrhage from the mouth. Neuffer's[50] patient died of a spontaneous hemorrhage from the mouth. Autopsy showed an aneurysmal dilatation of the internal carotid artery with an opening leading into the pharynx. Coomes,[13] Velebil,[65] Beck,[3] Booth,[6] Heaton,[2] Hirsch,[30] Klarfeld,[44] Stumpf, Tollet, and Ortmann lost their patients in the same way.

Generally speaking, the symptoms are frank and not, as some observers would lead us to believe, obscure. Cases similar to those just cited are the exception and not the rule. On inspection of the throat, the examiner sees a bulging in the lateral pharyngeal wall. The aneurysmal tumor may be circumscribed or it may be diffuse. To the touch it is soft and elastic, and pulsates throughout its entire extent. The swelling may, on occasion, so encroach upon the fauces as to convert the alimentary tract at this point into a narrow slit. With but rare exception the pharyngeal protrusion is accessible both to sight and to touch. Occasionally, however, the jaw is locked and the condition of the throat is neither visible nor palpable (Neuffer, Pircher). Externally there may be no evidence of the disease, or there may be a bogginess or a distinct lump behind the angle of the jaw (Porter,[56] Godfray,[26] Polak,[55] Jianu,[34] McMullen and Stanton,[46] Henle in Lebram, Stumpf, Blair in Brown, and Campbell. With a stethoscope placed over the swelling a bruit may be heard. Both murmur and pulsation disappear when the common carotid artery is compressed against the vertebral column, to reappear immediately on release of the pressure. Arrest of the circulation through the affected vessel causes a diminution in the size of the aneurysm. The patient may complain of annoying buzzing noises in the ear, persistent and severe hemicrania, vertigo and weakness. Swallowing of solids is sometimes impossible and is generally accomplished with difficulty. Liquid food may be regurgitated. Dyspnea is a common complaint.

It is a well recognized fact that aneurysm of the internal carotid artery rarely points in the neck. On January 22, 1808, Sir Astley Cooper[14] ligated the common carotid artery for what he believed to be an aneurysm of the

internal carotid artery. Inasmuch as the manifestation of the swelling was in the neck and not in the pharynx, the case has been classed by some writers as an aneurysm of the primitive trunk and not of the internal branch. In justification thereof, these critics assert that aneurysm of the internal carotid presents in the throat, never in the neck, while aneurysm of the common carotid occupies the neck, never the throat. They claim that in this peculiar behavior lies the chief difference between aneurysm of the internal carotid and one situated on the common trunk. Generally speaking, this is true. Yet at operation and at autopsy, instances of aneurysm of the internal carotid artery have been observed without any swelling having appeared in the throat. Prewitt,[57] Agnew,[1] Pircher, Sêrcer, Mensing and Regan, and Shipley, Winslow and Walker[60] have recorded observations of the kind.

On the other hand, isolated examples of aneurysmal disease of the common carotid artery have been observed in which the sac projected into the throat. Fehleisen,[21] in 1881, reported a case exhibiting this feature. The patient, a male, age 34, was taken ill April 2, 1879, with an attack of angina, which was followed by a quickly increasing tense swelling under the right jaw, accompanied by cyanosis and dyspnea. When admitted to the hospital April 7, the right half of the throat was red, swollen and definitely fluctuant. On April 9, von Bergmann incised the swelling and evacuated coagulated blood. Threatening asphyxia necessitated a tracheotomy, during the performance of which the patient died. Autopsy revealed, at the bifurcation of the common carotid artery, a perforation that emptied into a sac on the inner side of the artery. The sac was filled wih clotted blood and at its upper end was a rent which communicated with the pharynx. No pulsation was detected in the aneurysm nor was any bruit heard over it on auscultation.

Equally as fallacious is the contention of those who assert that aneurysm of the internal carotid artery always presents in the throat, as thoroughly reliable operative and necropsy findings have been adduced to the contrary (Porter, Edmunds,[20] Polak and Moser[49]).

The absence of cervical swelling is explained by the dense cervical fascia in front and the cervical vertebrae behind crowding the gradually dilating sac inwards toward the tonsillar fossa where the weak superior constrictor muscle and the mucous membrane interpose but slight resistance to the progress of the sac. Thus, the deep situation of the artery, covered as it is externally by the stylohyoid, stylopharyngeus, and the styloglossus muscles, and by dense aponeurotic tissues which extend to the styloid process, prevents any very marked prominence of such a tumor on the surface of the neck, and, as the artery is separated from the pharynx only by the mucous membrane and the constrictor muscle, it follows, as a matter of necessity, the lines of least resistance, pushes through the latter structures and bulges into the pharynx. A factor that also influences the point of appearance of this variety of aneurysm is the height at which the common carotid artery

bifurcates. If the carotidean bulb be situated at a high level, aneurysm of the internal carotid artery will point in the throat. When the bifurcation occurs low in the neck, aneurysm in the internal branch is apt to appear in the neck.

In isolated instances the pulse may be absent or so weak as to escape detection (Dubrueil,[18] Texier[62]). Johnson and Booth have each reported a case in which they were unable to detect any pulsation or to hear a bruit. Under such circumstances, the aspirating needle has supplied the clue to the true nature of the lesion (Texier, Perthes in Keppler,[53] Lyot et Petit,[43] Wulff, Zamboni in del Fabro, Cannuyt,[9] Hulbert[33] and Tollet). As diagnostic puncture leaves the aneurysmal wall weakened, aspiration should not be practiced unless the patient is so situated that a prompt operation can be performed if the sac should rupture through the path traversed by the needle. It should be remembered, however, that a dry tap does not preclude the possibility of aneurysm, as the point of the needle may be entangled in, or the bore of the cannula may be plugged with, clot. Instances of the kind have been recorded and the method is only of value when blood is obtained (Helferich in Werner[28]).

A recent diagnostic innovation of much promise is arteriography. In obscure vascular lesions visualization of the arterial tree has established itself as a procedure of merit. On at least two occasions, an extracranial aneurysm of the internal carotid artery has been detected by this method that might otherwise have been missed. We refer to the cases reported by Terry and Mysel, and by Tönnis. A puzzling feature of these two cases was the absence of a lump either in the throat or in the neck. Under the circumstances, arteriography proved absolutely necessary to clarify the diagnosis as well as to fix the exact location of the lesion.

Chronic unilateral swellings of the pharynx should be scrutinized with unusual care and especially so when the signs are lacking, obscure, atypical or bizarre, because a wrong diagnosis will almost certainly lead to mischievous surgery. Dubrueil considered an aneurysmal tumor in this situation as either an adenoma or a sarcoma of the tonsil and attempted its extirpation. During the progress of enucleation, the sac was torn. Ligation of the primitive carotid arrested the bleeding, but the patient eventually died of an hemiplegia. Helferich made the same mistake, but saved his patient by ligating the common carotid artery. Gross,[27] in an effort to drive home the lesson of a correct appraisal of the condition, cites a case recorded by Syme.[61] To stress his point, Gross naively adds: "Had a less careful surgeon had the management of the case, he might possibly have punctured the tumor and thus hurled the patient out of existence."

The differential diagnosis of lesions in this region is in the main between aneurysm in the extracranial portion of the internal carotid artery and peritonsillar abscess and is to be determined upon the same general principles that apply in differentiating these affections elsewhere. The history may

prove of much help in avoiding pitfalls. Mudd in Hulbert's case obtained a history of a lump antedating a scarlatinal angina and was thus led to a correct conclusion.

Agnew bases the making of a proper diagnosis on the ability of the surgeon both to see and to feel pulsation, but he fails to take into consideration the possibility of the patient being unable to open the mouth wide enough either for inspection of the throat or to permit the passage of a finger. Yet instances of the character have been recorded. The cases of Perier, Pircher and Neuffer serve to illustrate this point. However, failure to make a digital examination, when possible, is inexcusable.

Were this aneurysm more accessible, restorative aneurysmorrhaphy would be the ideal treatment. LeFort,[40] in a case of the arteriovenous type, incised the vein, sutured the hole in the artery and capped the line of suture with a section of the vein. The procedure proved successful. On the other hand, McMullen and Stanton lost the patient upon whom they performed an aneurysmorrhaphy. They thought that the operation had proved successful. The patient had been discharged from the hospital but had to return a few days later on account of the appearance of a hematoma beneath the scar. The wound was reopened and the source of the bleeding was found to be from a rent in the wall of the artery. Attempts to clamp the vessel failed owing to the friability of the tissues and the hemorrhage was controlled only with considerable difficulty by tamponing the wound. The woman had, however, lost so much blood that she soon died in collapse. This was an aneurysm of the arterial variety.

Morestin,[48] Moser, Bruns,[7] and Perthes[53] practiced extirpation of the sac. Agnew, because of recurrence of pulsation in the sac, ligated the opposite carotid, but the patient died about a week later of secondary rupture of the aneurysm into the mouth and of sepsis. Vander Veer[63] thought that he had obtained a cure by compression but the man fell dead three months later.

The operation of choice is ligation of the internal carotid artery proximal to the aneurysm. Should the internal carotid be unavailable for ligation, both the common and external carotid arteries must be tied along with any branches of the latter arising between the site of the ligation and the bifurcation. The latter provision prevents a possible reëstablishment of the circulation in the sac by a reversal of the current through the open segments of the external and common carotids. Failure to observe this precaution explains the reappearance of pulsation in the sac.

Before permanently arresting the circulation in the internal carotid artery, the primitive trunk should be temporarily occluded under local anesthesia, to forestall the evil results incident to a disturbed cerebral circulation. Usually a defective circulation will manifest itself promptly by vertigo and by faintness. In the face of these warnings, the ligature should be removed without delay.

Competent authorities are agreed that ligation either of the common trunk or of its internal branch is not an operation to be undertaken lightly. Johnson[36] says that paralysis occurs in 15 per cent of the cases. According to DaCosta,[17] in from 20 to 25 per cent of the cases there is cerebral softening or some other intracranial mischief after ligation of the common carotid artery. Crile[16] states that, of the cases developing cerebral trouble, one-half of the patients die. The direct operative mortality according to him is 3 per cent. Horsley[32] asserts that the danger associated with ligation of the common carotid artery increases enormously after 40 years of age and is due to the diminished blood supply to the brain. On the other hand, he feels that ligation of the internal or of the common carotid in the young with elastic arteries is comparatively free from danger. In the opinion of this surgeon, cerebral symptoms vary from giddiness to complete hemiplegia.

As a security against undesirable symptoms, Jordan,[37] of Heidelberg, recommends a procedure which proved itself of value in an operation for carcinoma of the neck; namely, previous loose constriction of the carotid for 48 hours. By carefully constricting the carotid with a small, flat piece of tape until the peripheral pulse just ceases, there is no injury to the intima and no formation of clot. If the ligature be removed after two days, the peripheral pulse reappears and soon attains its normal strength.

Preliminary ligation should be performed under local anesthesia, so as to obtain prompt information of any effect upon the cerebrum. If disturbances occur after the constriction, the ligature is removed from the artery through the wound, which has been left open, and the circulation restored to normal.

By gradually tightening the ligature, an adequate collateral circulation may, on occasion, be developed from what was an inadequate one. This is also the teaching of Halsted, and is based on the toleration of the carotid to temporary occlusion. Crile has demonstrated that the common carotid artery may be clamped for 48 hours without permanently damaging the intima or causing thrombosis. Halsted employs aluminum bands for this purpose. It must be remembered, however, that cerebral symptoms may occur one or two weeks after ligation of these vessels. Matas[45] says: "In the light of this important suggestion, no one is justified in planning a deliberate operation for the cure of aneurysm in this perilous locality without first testing the efficiency of the collateral circulation. The possible occurrence of cerebral disturbances has invested a simple technical procedure with a gravity associated with but few operations."

In passing we desire to report that Winslow's patient remains completely cured. It is now more than 14 years since the operation. The woman is enjoying good health and is working daily as a cook. The completed history of the case reported by Shipley and Lynn,[60] in 1916, is also available. The patient, a white married woman, age 52, was admitted to the University Hospital for the last time January 7, 1926, and was discharged January 24,

in an unimproved condition. Eleven years before, her left common and external carotid arteries had been tied for an extracranial aneurysm of the internal carotid artery. The aneurysm had not grown since then but had shrunk somewhat in size. The patient, however, was being disturbed by noises in her head, throbbing sensations in her throat and severe headache. Active pulsation was felt in the position of the primitive carotid over the point of the former ligation. Shipley, therefore, decided to explore the site of the old operation to ascertain whether anything had happened at the place where the carotid had been tied. It was thought that an artery was probably entering the sac from below and an operation was performed January 8, 1926, for the purpose of ligating this artery. However, the artery was not tied because it apparently did not communicate with the sac. The patient died in the summer of 1927, of a right-sided hemiplegia.

These results would indicate that permanent arrest of the circulation through the internal carotid artery has little effect on the longevity of life in those individuals who have weathered successfully the initial storms incident to such an operation.

Aneurysm in this situation is either true or false. The true is that variety in which either one or all of the tunics of the artery enter into the composition of the sac. The false is that type in which the wall of the sac is formed by adventitious tissue. According to its etiology, this aneurysm may be classified clinically as (1) spontaneous, (2) erosive, and (3) traumatic. In the traumatic variety the artery alone may be the seat of the aneurysm or the artery and its satellite vein may be involved in the process.

No matter what the type, all of these aneurysms give rise to practically the same symptoms. They tend to present as a pulsatile mass in the lateral pharyngeal wall and require some kind of radical vascular surgery for their cure.

The prognosis after operation is good. If these patients are left, however, to the beneficent mercies of nature, death will ensue sooner or later. Thus, in Winslow's collected series of 1926, 70 per cent of the patients operated upon since 1900 were cured, whereas, only 10 per cent of the patients not operated upon recovered. Peculiarly enough, all of the latter patients, two in number, were under the care of one man—Castex.[10]

We realize thoroughly that the percentage of recoveries is, in all probability, too high, for one is prone to report successes and to forget failures. Withal, when these figures are pitted the one against the other, there can be no question of the manifold advantages of operation over a *laissez faire* policy.

Case Reports

Case 1.—A colored man, age 38, married, was admitted to the University Hospital November 6, 1932, with a pulsating lump in the left side of the neck. He had been shot with a bullet August 12, 1932. The ball entered the back of the neck an inch to the left of the midline on a level with the lobe of the ear to emerge one and one-half inches below the mandible and an inch to the left of the midline. The swelling,

which developed immediately, subsided for a time but at the end of two weeks began to increase slowly in size and to ache.

Examination revealed a slightly compressible tumor which expanded and beat in unison with the heart. The swelling harbored an audible murmur and a palpable thrill. The bruit and the fremitus lasted throughout systole and diastole but were more pronounced during systole. The lump was oval in shape and the size of an orange. It filled the left submaxillary triangle and encroached upon the superior carotid and posterior cervical triangles. There was no swelling in the tonsillar region. Pulse 78; respirations 20; temperature 98.3° F.; blood pressure 120/70. The physical examination was negative with the exception of a penile sore for which the patient had received antiluetic treatment. The aneurysmal nature of the lump was evident, but the exact vessel involved was in doubt. It was thought that the lesion was most probably an arteriovenous fistula between the external or the internal carotid artery and a neighboring vein.

FIG. 1.—(Case 2) Drawing showing the appearance of the protruding mass in the tonsillar fossa. (The artist has reversed the position of the tumor.)

On November 9, 1932, under avertin and supplementary ether anesthesia, an incision was made along the anterior border of the left sternocleidomastoid muscle. The common carotid artery was exposed and dissected free up to its bifurcation. In doing this, it was necessary to ligate the facial vein and the superior thyroid artery and vein. After considerable difficulty, the external carotid artery, which was densely adherent to the aneurysm, was isolated and ligated just beyond the bulb of the primitive carotid. This maneuver did not stop pulsation in the sac. The internal jugular vein was accordingly exposed but was not found to be dilated, which fact ruled out a communication with the artery. To arrest the circulation in the aneurysm, the common carotid artery had to be ligated also. The postoperative course was uneventful. At no time did any cerebral complications develop. The patient was discharged from the hospital December

5, 1932, as cured. The pulsation, thrill, murmur and expansion in the swelling had ceased, and the mass had diminished to one-half of its original volume.

Unlike most extracranial aneurysms of the internal carotid artery this one did not produce a bulging in the throat, but caused a swelling in the neck.

Case 2.—The patient, a white male, age 27, married, was admitted to the University Hospital May 2, 1936, with a mass in the left side of the pharynx which had been diagnosed as an extracranial aneurysm of the internal carotid artery. The lump was discovered by his physician during a routine physical examination two months before. In April, the patient noticed that the swelling was increasing in size. His tonsils had been removed when he was seven years of age.

Inspection revealed a pulsating mass in the left tonsillar fossa. The tumor was the size of a robin's egg and protruded inward (Fig. 1). No bulging was seen in the neck. The swelling expanded in all directions. Pulsation in the mass stopped on compression of the corresponding common carotid artery against the vertebral column to begin anew with the release of the pressure. The physical findings were otherwise of no moment. The Kolmer test was negative on two occasions, as were also the routine laboratory examinations. In the absence of any discoverable cause, the aneurysm was considered of spontaneous origin.

On May 12, 1936, under gas and ether anesthesia, a linear incision was made along the anterior margin of the left sternocleidomastoid muscle. The carotid sheath was opened and the common, external and internal carotid arteries were exposed. The external carotid was displaced laterally and two silk ligatures were placed around the internal carotid just above the bifurcation. The ligatures were not tied until traction upon them had clearly demonstrated cessation of pulsation in the sac.

The postoperative course was smooth and the patient was discharged from the hospital June 2, 1936, as cured. The swelling had shrunk considerably and did not pulsate. The operation caused no cerebral disturbances.

COMMENT.—This study is based on 18 cases of extracranial aneurysms of the internal carotid artery, of which 16 were culled from the current literature and the two observations herewith presented (Table I). Six of these aneurysms were spontaneous in origin; two, traumatic; nine, erosive, and one was of the arteriovenous type. Extracranial aneurysm of the internal carotid artery occurs in about an equal ratio between the male and the female sexes. In this series, nine of the patients were males and eight were females. In the remaining case the sex was not noted. The right side was affected nine times and the left side nine times. Of the 18 patients, ten, or 55.56 per cent, were cured; two, or 11.11 per cent, showed no improvement, and six, or 33.33 per cent, died. Aneurysm in this situation is a disease of youth. Of the series, 15, or 83.33 per cent, were under 40 years of age. The youngest patient was 11 months old and the oldest 61 years. The cases were distributed in decades as follows: Three in the first; four in the second; three in the third; five in the fourth; one in the sixth, and one in the seventh. One case was described as that of a woman. Some type of radical vascular surgery was performed on 12 patients, with ten, or 83.33 per cent, recoveries, and two, or 16.67 per cent, deaths (Table II). Conservative treatment was practiced on six patients. Two, or 33.33 per cent, of these patients were unimproved and four, or 66.67 per cent, died (Table III).

TABLE I

SUMMARY OF THE 18 CASES HEREWITH REPORTED UPON

Type	Cured	Unimproved	Died	Total Cases
Spontaneous	5 (83.33%)	1 (16.67%)	0	6
Erosive	3 (33.33%)	0	6 (66.67%)	9
Traumatic	1 (50.0%)	1 (50.0%)	0	2
Arteriovenous	1 (100.0%)	0	0	1
Total cases	10 (55.56%)	2 (11.11%)	6 (33.33%)	18

TABLE II

SUMMARY OF THE 12 CASES OPERATED UPON

Type	Cured	Unimproved	Died	Total Cases
Spontaneous	5 (100.0%)	0	0	5
Erosive	3 (60.0%)	0	2 (40.0%)	5
Traumatic	1 (100.0%)	0	0	1
Arteriovenous	1 (100.0%)	0	0	1
Total cases	10 (83.33%)	0	2 (16.67%)	12

TABLE III

SUMMARY OF THE SIX CASES TREATED CONSERVATIVELY

Type	Cured	Unimproved	Died	Total Cases
Spontaneous	0	1 (100.0%)	0	1
Erosive	0	0	4 (100.0%)	4
Traumatic	0	1 (100.0%)	0	1
Arteriovenous	0	0	0	0
Total cases	0	2 (33.333%)	4 (66.667%)	6

The diagnosis should offer no great difficulty if one bears in mind the possible occurrence of the lesion. On occasion, however, it may mimic a tonsillar abscess. Under such circumstances, extreme care must be exercised to avoid a wrong diagnosis. The prognosis is good in those cases subjected to operation, but the outlook is bad for those conservatively treated

or left to the beneficent influences of nature. Nonoperative treatment has proved worthless. In this small series, 66.66 per cent of the patients so treated died. Of the others, one refused to be operated upon and left the hospital against advice. She could not be located at the time of Robert's report. In the second case, Tönnis believed an embolus had been washed off the sac and had lodged in the anterior cerebral artery. Since the aneurysm itself was inaccessible to operative attack, the only alternative treatment was ligation of the internal carotid artery. Tönnis rejected the latter procedure because of the possibility of increasing the cerebral damage. The diagnosis in this case was made by arteriography. The report did not mention either a pharyngeal or a cervical swelling as being present. The patient was a paralytic. The operation of choice is ligation of the internal carotid artery. The following ligations were performed in this series:

Ligation of the internal carotid artery	3
Ligation of the internal carotid artery, common carotid, internal jugular vein, lingual and facial veins	1
Ligation of the common carotid artery and internal jugular vein	2
Ligation of the common carotid, sac incised and packed	1
Ligation of the common carotid artery	1
Ligation of the common carotid artery by strangulation	1
Ligation of the common carotid artery by fascial strips	1
Ligation of the common carotid, external carotid, superior thyroid arteries and facial and superior thyroid veins	1
Ligation of the common carotid, external carotid, superior thyroid, lingual and facial arteries and sac opened and packed	1

Conservative therapy has nothing to offer in the way of cure and should be tabooed. Those who employ it court disaster. The story unfolded in the reports is a narrative of sad catastrophes. Six cases in this series were treated conservatively without a single cure. Two died of spontaneous rupture of the sac into the mouth, one of an uncontrollable hemorrhage from an incision into the sac, and one of an ulcerative endocarditis. Two of the patients left the hospital with the lesion in the same condition as when they entered.

It is true that Castex[10] reports the cure of two patients by nonoperative measures. In both of these cases, the patients had syphilis. Castex cured these aneurysms by antiluetic medication and local applications. No other reported cases were benefited in the least by such treatment. In view of the high mortality rate attached to conservatively treated cases, some form of radical arterial surgery must remain our sheet anchor in the future as in the past, in spite of the gratifying results obtained by Castex.

The cases of Tollet and Ortmann are grim reminders that death ever threatens from spontaneous rupture of the sac into the mouth, when appropriate surgical measures are withheld.

The nonoperative methods of treatment employed in this series were as follows:

Supposed abscess incised, no pus, only blood escaped	1
No treatment: (1) Ligation rejected on account of possibility of increasing cerebral damage	1
(2) Patient had ulcerative endocarditis	1
Compression of common carotid artery	1
Spontaneous hemorrhage, in hospital from January 12 to February 10, could not inspect throat because jaws were locked	1
Patient would not permit a ligation of the carotid	1

CONCLUSIONS

(1) Extracranial aneurysm of the internal carotid artery is not a common condition, but it occurs often enough to be borne in mind when one is dealing with unilateral faucial swellings.

(2) It is not so much the rarity of the affection that commands attention as its propensity to imitate peritonsillar abscess, which habit of mimicry has, on occasion, led to its being lanced, with fatal results. Therefore, it is much the better policy to look, feel and listen before cutting a bizarre unilateral swelling of the throat than to bemoan at leisure a hasty act.

(3) Spontaneous cure may occur, but the usual termination in untreated cases is death from rupture of the sac into the patient's mouth.

(4) Ligation of the internal carotid artery is the treatment of choice. If this is impossible, then occlusion of the common carotid artery together with ligation of the external carotid artery between its origin and its first branch should be practiced. If the external trunk be tied distal to a branch, the branch must be ligated also. This provision forestalls the possibility of a reestablishment of the circulation through the internal carotid artery.

(5) The majority of patients afflicted with the lesion should recover, if the aneurysm be promptly recognized and properly treated, but the mortality rate will be high under blundering, dilatory, or pernicious tactics. After ligation, the prognosis is good both as regards operative recovery and permanent cure.

(6) Aneurysm in other situations is far more prevalent in the male than in the female; in the internal carotid artery it occurs in almost an equal ratio in the two sexes, being slightly more prevalent in the male if all types are considered, but more frequent in the female in the spontaneous variety.

Those interested in a further study of the subject may consult the appended tables. Therein are tabulated the essential features of the 18 cases cases composing this series (Table IV). For the sake of completeness we have summarized in Table V ten additional cases in which the descriptive matter accompanying them does not offer sufficient evidence to warrant a positive statement as to their actual character. All have a distinct bearing, however, on the question of extracranial aneurysm of the internal carotid artery and merit notice. In order to conserve space, we present them in a supplementary table labeled "Doubtful Cases" (Table V).

TABLE IV

SUMMARY OF THE CASE HISTORIES OF THE 18 PATIENTS COMPOSING THIS SERIES

Case	Author	Sex Age Side	Type Duration	Operation or Treatment Date	Cured	Died	Remarks
1	Henle in Lebram, P.[1]	F. 9 R.	Erosive 1 week	Ligation right common carotid artery, and internal jugular vein; later because it was wounded during operation Dec. 2, 1902	Yes	...	Admitted, Dec. 1, 1902, with fever of 14 days' duration. For 3 days bleeding from mouth, nose and right ear. In right side of neck below jaw was a nonfluctuating, tender lump. Soft palate was bulged on right side by a tumor with indistinct fluctuation. Swelling reached across midline. Incision liberated coagulated blood mixed with pus. Night of Dec. 2/3, patient bled freely from mouth. Paralysis of left side developed after operation. Discharged, Jan. 3, 1903, with swelling much smaller.
2	Stumpf, H.[2]	F. 5 R.	Erosive 1 week	Ligation right common carotid artery Oct. 18, 1915 Religation common carotid, strongly pulsating jugular vein also tied Oct. 25, 1915	...	Yes	Sick 14 days with painful swelling in neck, and for 4 days a bloody discharge from right ear. Oct. 15, 1915, lump was seen behind ascending ramus of jaw bone. In right side of throat was a resistant swelling. Tonsil bulged inward. Oct. 18, free bleeding occurred from right ear and from nose. Oct. 25, hemorrhage recurred from ear, nose and mouth. Patient died 5 minutes after second intervention. Autopsy: dilated and spindle-shaped right internal carotid artery near its entrance into carotid canal. Artery had eroded into cavity of an abscess.
3	Reid, M.[3]	F. 61 R.	Spontaneous 1 year	Ligation right internal carotid artery Sept. 2, 1919	Yes	...	White woman, married, admitted, Aug. 27, 1919, for extracranial aneurysm of internal carotid artery. No history of lues and negative Wassermann test. Trouble began with tickling sensation in throat. Later, a swelling resembling a peritonsillar abscess protruded into fauces. Eight weeks before admission an incision had been made into swelling. Mass was pulsatile and involved soft palate and tonsil. It did not show externally. For 8 days, patient had postoperative right hemicrania. At no time was there any sign of paralysis. May 29, 1922, patient felt a little fulness in throat which, however, did not pulsate.
4	Blair, V. P., in Brown[4]	F. 36 L.	Spontaneous 7 years	Ligation left common and external carotids, superior thyroid, lingual and facial arteries; swelling opened and packed with gauze June, 1926	Yes	...	Swelling appeared under angle of jaw in 1919. It was round, smooth, 5x4 cm., pulsatile and harbored a bruit. Tongue was pushed to right side by swelling. Patient complained of dysphagia; had had 3 apoplectic seizures; was married, and had 2 children who were living and well. Wassermann test was negative. In Dec. 1926, the woman appeared to be cured. She was at that time 4 months' pregnant.

5	Sêrcer, A.[5]	F. 22 L.	Erosive 2 weeks	Ligation left common carotid artery, blood continuing to escape from wound; aneurysmal sac packed with gauze; bleeding then ceased; local anesthesia Nov. 24, 1927	...	Yes	Admitted, Nov. 24, 1927, with angina of 5 weeks' duration. Left side of neck abscessed and was incised. Day before admission—15 days after incision of abscess—patient vomited clotted blood. Beneath angle of jaw was lump, size of man's fist. Patient repeatedly spat blood. On third postoperative day, hemiplegia developed. Patient died 4 weeks after operation. Autopsy: abscess in left hemisphere; site of erosion in internal carotid artery not found macroscopically, only on section. Sêrcer called it a spurious aneurysm. False aneurysm consecutive to a tonsillar abscess.
6	Thornval[6]	M. 19 L.	Erosive	Ligation common carotid artery Sept. 2, 1928	Yes	...	
7	Tollet, W.[7]	M. 18 R.	Erosive 1 month	Ligation right internal carotid artery Aug. 9, 1929	Yes	...	Illness began, June 10, 1929, with angina followed by peritonsillar abscess which was incised, June 19. Following day free hemorrhage ensued, which ceased spontaneously. Six weeks after onset of illness, patient returned with pain in neck. For past 4 days he had had dysphagia and tinnitus in right ear. Inspection of pharynx revealed protrusion above and lateral to tonsil. Palpation of swelling disclosed pulsation, and on puncture the syringe withdrew arterial blood. Diagnosis evident. Discharged 4 days postoperatively. In a few weeks, pharyngeal swelling had disappeared. No signs of lues. Nov. 12, 1929, cure maintained itself.
8	Mensing, E. H., and Regan, J. R.[8]	M. 36 L.	Spontaneous 1 year	Ligation left common carotid artery by gradual strangulation Mar. 6 to Mar. 15, 1930	Yes	...	White man, admitted, Feb. 4, 1930, with lump in neck. It appeared in Aug., 1929, had slowly increased in size but had caused no discomfort. Questionable history of chancre in 1918. Had not received luetic treatment. Two inches below lobe of ear was pear-shaped mass, of ovoid form, with poles in line of sternomastoid muscle and expansile pulsation. Wassermann reaction, Feb. 8, doubtfully negative; recheck, Feb. 14, negative. Diagnosis: Aneurysm of internal carotid artery. Operation revealed a sacular aneurysm. June 30, 1930, patient in best of health, but 2 weeks before swelling somewhat smaller than hen's egg had appeared in right side of neck. Mass animated by expansile pulsation. Diagnosis: Aneurysm of right internal carotid artery.
9	Campbell, J. L.[9]	F. Adult L.	Spontaneous 2 years	Partial occlusion with an autogenous fascial band 1933	Yes	...	Mulatto woman, admitted with diagnosis of peritonsillar abscess. For 2 years had noticed a throbbing lump in neck just below ear. Night before admission, she spat blood. She could scarcely open her mouth, swallowed with difficulty and spoke in a whisper. Left tonsil pushed well beyond uvula; soft palate swollen and red. While preparations were being made to open supposed abscess, aneurysm discovered. Patient left hospital much improved.

TABLE IV (*Continued*)

Case	Author	Sex Age Side	Type Duration	Operation or Treatment Date	Cured	Died	Remarks
10	Terry, T. L., and Mysel, P.[10]	M. 38 R.	Arteriovenous	Ten days after injection of thorium, wound was reopened under general anesthesia and whirling pools and eddies of arterial blood were seen in internal jugular vein; jugular and lingual veins ligated; facial vein tied at time of injection of thorium. Tissues about internal carotid artery for 1½ inches above bifurcation were boggy. To prevent back-wash from external carotid, common trunk ligated and internal branch tied beyond infiltrated area 1934	Yes	...	Three weeks after onset of pain in right temporal region, patient noticed a swishing sound in right ear; also vertical diplopia which soon disappeared. Noise became so intense as to prevent work. Hit on head with ax 20 years before and 2 months ago struck in temple by piece of timber. Examination: slight exophthalmos and pulsation of right eye, latter synchronous with radial pulse; superficial veins more prominent in right side of neck; loud, shrill, musical murmur continuous but greatly accentuated during systole heard over right eye, in temple and neck. Compression of carotid stopped bruit but had no effect on exophthalmos. Conjunctival vessels of right eye increased in size and tortuous; fundal veins moderately dilated and more tortuous than normal; retinal arteries normal in size and distribution; no retinal hemorrhages or edema. Roentgenogram of skull showed no increased density in orbit, or fracture. Wassermann test negative. Symptoms were against aneurysm being in or just behind orbit. To determine its exact location, internal carotid exposed and 25 cc. of thorium dioxide solution injected into it. Roentgenograms during and after injection showed a triangular shadow with base near site of injection and apex just below jugular bulb. None of thorium entered skull. Findings proved that a fistula existed between internal carotid and internal jugular in upper part of neck. Day after operation, pulsation of eyeball, tinnitus, headache and bruit had disappeared. No postoperative cerebral complications developed. Five months after operation, exophthalmos was still present. Authors thought aneurysm of spontaneous origin and not due to old or to more recent cranial injury. As far as they could ascertain, this is the only reported case of pulsating exophthalmos caused by an arteriovenous aneurysm in the neck.
11	Shipley, A. M., Winslow, N., and Walker, W. W.[11]	M. 38 L.	Traumatic 3 months	Ligation left common carotid, superior thyroid, external carotid arteries, and facial and superior thyroid veins; avertin anesthesia supplemented by ether	Yes	...	Negro, admitted, Nov. 6, 1932, with aneurysm of left internal carotid artery caused by bullet wound received on Aug. 12, 1932. Swelling developed immediately but subsided for a time. At end of two weeks, lump began to increase slowly in size and to ache. The slightly compressible tumor expanded and beat in unison with heart, and had an audible murmur and a palpable thrill. Fremitus and

				Nov. 9, 1932			bruit lasted throughout systole and diastole, but were more prominent during systole. Swelling was egg-shaped, oval, size of an orange. It filled left submaxillary triangle and encroached upon superior carotid and posterior cervical triangles. Gave history of penile sore for which he had received antiluetic treatment. Mass pointed in neck and unlike most aneurysms in this situation did not produce a bulging in tonsillar region. Discharged, Dec. 5, 1932, as cured. Pulsation, thrill, murmur and expansion in swelling had ceased, and mass had diminished to two-thirds its original volume. At no time did any cerebral symptoms develop.
12	Shipley, A. M., Winslow, N., and Walker, W. W.[12]	M. 27 L.	Spontaneous 2 months	Ligation left internal carotid artery May 12, 1936	Yes	...	White man, married, admitted, May 2, 1936, with an expansile, pulsating mass, size of a robin's egg, in left side of pharynx. No bulging seen in neck. Pulsation stopped on compression of common carotid artery. Kolmer test negative on 2 occasions. Operation caused no cerebral disturbances.
13	Stumpf, H.[13]	M. 13 L.	Erosive 2 weeks 1915	None	...	Yes	Illness began, Jan. 25, 1915, with angina. Feb. 2, left-sided peritonsillar abscess developed. Admitted, Feb. 3. Left tonsil and uvula pushed to right, swollen and red. On incision no pus escaped, only blood. During night of Feb. 4, patient spat blood. Incision again yielded blood. Cavity packed. Tampon removed, Feb. 6. That night active hemorrhage began and child died before any treatment could be given. Autopsy: Erosive aneurysm of left internal carotid artery, size of pea, in cavity of peritonsillar abscess.
14	Tönnis, W.[14]	M. 36 L.	Traumatic 1½ years 1918	None	Same	...	Exploding shell, in 1918, caused multiple wounds, among them one in neck. After recovery, patient well until July, 1932, when he experienced fleeting weakness in right arm. In Feb., 1933, oculist consulted for failing vision in left eye. Mar. 29, 1933, had another attack of paralysis in right arm along with loss of speech. Both quickly subsided. Sept. 19, 1933, paralysis recurred and he felt severe pain in head. Ventriculography revealed nothing abnormal, so Tönnis decided to perform arteriography. Sept. 29, 1933, local anesthesia, left common carotid artery isolated at its bifurcation and 6 cc. of thorotrast injected into lumen of internal branch. Roentgen ray films showed aneurysm, size of bean on internal carotid artery in front of and a finger's breadth to left of first and second cervical vertebrae. Ramifications of internal carotid visible but trunk of anterior cerebral could not be identified. In this region were many fine, threadlike vessels. Paralysis attributed to embolus having been washed from sac to lodge in anterior cerebral artery. Since aneurysm was inaccessible to operative attack, the only alternative treatment was ligation of internal carotid artery. Latter procedure rejected because of fear of increasing cerebral damage.

TABLE IV (*Continued*)

Case	Author	Sex Age Side	Type Duration	Operation or Treatment Date	Cured	Died	Remarks
15	Tollet, W.	.. 11 mos. R.	Erosive 1 month	None 1926	...	Yes	Unconscious infant admitted, Dec. 30, 1926, with blood escaping from right ear and throat filled with coagula. Although bleeding ceased on compression of carotid and tamponade of ear and pharynx, child died in a few minutes. Mother had noticed, on Dec. 25, swelling beneath child's ear. Dec. 29, bloody discharge began to flow from ear. A doctor on attempting to remove clot from auditory canal started up a violent hemorrhage which soon ceased. In night bleeding recurred, to stop spontaneously. On repetition of hemorrhage next day, child was brought to hospital. Autopsy: Marked bulging at upper part of right lateral pharyngeal wall with no detectable perforation; internal carotid artery thickened for 5 mm. of its length to a point 1 cm. below its entrance into carotid canal. In median wall of involved area was a hole 1 Mm. in diameter. Cloaca opened into plum-sized cavity filled with clots. Wall of cavity was composed of a few millimeters thick membrane. At lower part of sac was a pea-sized hole which opened into eustachian tube near its pharyngeal end. No pus found. Sections of carotid at level of carotid foramen showed normal walls, but in neighborhood of rupture, adventitia and tunica media thickened by inflammatory products. Sac composed of connective tissue with areas of cellular infiltration—chiefly polymorphonuclear leucocytes. No part of arterial wall was seen in capsule. Case of pseudo-aneurysm caused by erosion of internal carotid artery.
16	Bullrich, R. A., y Besimsky, M.[16]	M. 26 R.	Erosive 1½ months	None 1927	...	Yes	Patient with ulcerative and vegetative endocarditis developed aneurysm of internal carotid near bifurcation of common carotid artery. Man died, July 31, 1927, or 1½ months after onset of symptoms. Autopsy: Fusiform aneurysm of internal carotid artery.
17	Ortmann, K. K.[17]	F. 19 L.	Erosive	None 1929	...	Yes	Perforation of internal carotid artery consecutive to cervical phlegmon with formation of false aneurysm in pharyngeal space and spontaneous rupture into nasopharynx, resulting in fatal hemorrhage. Admitted, Jan. 12, 1929; died, Feb. 10, 1929. She bled repeatedly from nose. Fauces could not be satisfactorily inspected because mouth was locked. Swelling in neck. Feb. 5, developed a right-sided hemiplegia. Autopsy was performed.

18	Roberts, A. D.[13]	F. 53 R.	Spontaneous 1½ years	None 1930		Same	...

White female, married, multipara, seen in Apr., 1930, with swelling in throat which had been gradually getting larger for 18 months. At times, woman felt throbbing in lump but never any pain. On looking into throat, one saw a rounded mass which filled whole right tonsillar area. Pulsation felt in tumor and a blowing sound heard with stethoscope placed on lump. Externally nothing was seen, felt or heard. Repeated and provocative Wassermann tests, as well as roentgenologic examinations, were negative. Pulse 114; blood pressure 184/94. Before ligation of common carotid artery could be performed, patient left hospital against advice and at time of report could not be located.

TABLE V

SUMMARY OF THE CASE HISTORIES OF THE TEN PATIENTS IN WHOM THE TYPE OF THE ANEURYSM WAS CONSIDERED DOUBTFUL

Author	Sex Age Side	Type Duration	Operation or Treatment Date	Cured	Died	Remarks
DeWar, D.[1]	F. 27 R.	....	Ligation common carotid artery June 2, 1859	Yes	...	Admitted, Apr. 8, 1859, with swellings about angle of jaw and lump in situation of tonsil which was supposed to be an abscess. External masses disappeared, but internal tumor continued to increase in size. On 3 occasions, profuse hemorrhages took place from it. Distinct pulsasion felt in lesion. Seven months after ligation, aneurysm had disappeared.
Lefort in Lefort, R., et Piquet, J.[2]	M. 52 L.	....	Periarterial sympathectomy, encirclement of vessel with strip of fascia; ligation and division thyro-linguo-facial trunk; dilatation involved 3 carotids, but especially bulb Oct. 14, 1925	Yes	...	In Aug., 1925, patient consulted Piquet for dysphagia. Examination: dilatation in carotidean bulb. On palpation, a vertical, elongated, violently pulsating, tender, ovoid mass, size of nut, felt. Bulgings and beats visible to sight. Patient also perceived them and was incommoded by them. Mass was reducible on compression. It had not the appearance of an arteriovenous fistula because it had no thrill, at most a slight systolic fremitus. Wassermann test on 2 occasions negative. Summary: One witnessed the development of a dilatation on the carotid bulb of rapid growth. In Apr., 1926, patient well, circulation normal, and dilatation had diminished.
Lefort in Lefort, R., et Piquet, J.[3]	F. 57 L.	..... 2 months	Ligation and division thyro-linguo-facial trunk; carotids were dilated but dilatation involved mostly internal carotid artery; periarterial sympathectomy, aponeurotic graft slid behind bulb of carotid and its margins sutured together in front of artery Dec. 12, 1927	Yes	...	Patient noticed a small pulsatile tumor in midcarotid region. This was a small arterial dilatation at level of carotid bulb. It formed a vertically elongated mass conserving the general form of the vessels. It was animated by beats; reducible on compression, which procedure produced uneasiness. No evidence of lues. Wassermann test negative. In Feb., 1929, pulsation in carotid artery visible, but expansion not abnormal. Mass scarcely more voluminous than opposite artery.
Duval, A.[4]	M. Man L.	Arteriovenous	Compression direct and digital pressure on common carotid artery 1862	Improved	...	Cut behind ear with knife. Admitted to hospital 8 hours afterwards with swelling in neck which harbored both bruit and thrill. Compression applied to carotid stopped murmur and fremitus. In first 10 days, 4 hemorrhages occurred. These were arrested by direct compression upon tumor and digital pressure on common carotid. Noise in tumor sometimes interfered with sleep. Feb. 15, 1864, bruit was less intense.
Franklin, M. M.[5]	.. 7	Erosive	Bleeding stopped by sponge pulled into and lodged in vault of pharynx	Yes	...	Admitted, May 11, 1896, for angina. Next day child found in syncope with blood flowing freely from nose and mouth. Examination: Pulsat-

	L.		by string attached to nasal catheter 1896			ing tumor in neck. Inspection of mouth disclosed blood gushing from a point posterior to tonsillar pillar. Hemiplegia of right side, with aphasia. Swelling in neck slowly disappeared. Nasal plug removed in 3 days. Year later, child had recovered speech and suffered only from slight disability of right arm and talipes equinovarus. According to Stump, this was an aneurysm of internal carotid artery. Data are too scanty to warrant its acceptance though it might possibly have been one in spite of its spontaneous disappearance.
Lebram, P.[6]	M. 2 R.	Erosive 2 weeks	None 1903	...	Yes	Admitted, Jan. 7, 1903. Cervical nodes swollen. Blood and pus were escaping from right ear. Right soft palate swollen, but not pulsating. Right side of neck incised but no pus found. Swelling in soft palate increased in size and encroached upon right half of throat. Jan. 12, 1903, swelling incised for peritonsillar abscess with escape of blood. Compression at site of cut and on carotid had no effect. Child died in 2 minutes.
Worms, G., et Germain[7]	M. Soldier R.	Arteriovenous 11 years	Injection of 25 cc. gelatin subcutaneously 1926	Improved	...	Wounded in neck, Feb., 1915, by piece of shell. When shock had disappeared, he felt a systolic souffle, synchronous with cardiac beats. After convalescence, man resumed his occupation and engaged in sports without inconvenience. He saw normally and was not bothered by his fremitus until 1926, when a severe fatigue was followed by cephalgia and nonpulsatile exophthalmos of right eye. In Aug., 1931, symptoms had not abated. Examination: At site of wound thrill, but no expansion. Similar fremitus felt in pharynx behind posterior pillar which pulsated synchronously with pulse. In parotid region continuous bruit heard. Pressure on common carotid artery stopped souffle and thrill. Cardiovascular system not affected. Diagnosis: Arteriovenous aneurysm of right internal carotid artery and internal jugular vein. As the condition had been stationary for 4 years, authors decided to ligate only in case of aggravation of symptoms.
Barnes, E. B.[8]	M. 7 ..	..... 2 years	None 1927	...	...	Seen, Apr. 5, 1927, for dysphagia. Fusiform aneurysm of internal carotid artery seen with laryngeal mirror. Author did not tie vessel because of patient's age, the probable condition of the fellow vessel which might not give an adequate supply of blood to brain, and the fact that patient had had condition for a long time and was fairly comfortable. Neither H. Thomas nor A. Wylie thought swelling to be an aneurysm but probably an ectasia.
McIntyre, D. R.[9]	M. 54 L.	....	None 1928	Same	...	White male, seen Mar. 6, 1933, for blowing noise in ear. Noise began in Feb., 1928. He had just recovered from otitis media on right side. Left side of neck swollen. Palpatation revealed no abnormal pulsation but on auscultation, a blowing, hissing sound was heard. It extended from below tip of mastoid process to top of left side of head. Blood pressure 180/90. Heart normal. If collateral circulation improved, author intended to tie carotid artery.

TABLE V (*Continued*)

Author	Sex Age Side	Type Duration	Operation or Treatment Date	Cured	Died	Remarks
Markowicz, H.[10]	M. 65 R.	..... 7 weeks	None 1933	Same	...	Lues at 21. Seven weeks before, pulsation in right side of head was so perceptible as to awaken him when sleeping. Blood Wassermann test positive 2 weeks before. Mar. 28, 1933, pulsations in both sides of neck and temples were very prominent. Had a slight Romberg. Tonsil: Posterior arch of palate and pharyngeal wall on right side pulsated actively. Posterior rhinioscopy revealed bluish-gray protrusion shimmering through mucous membrane on right side between nose and opening of eustachian tube. It was as broad as a finger and 2 cm. long. Its walls seemed extraordinarily thin. If vessels of neck were compressed, pulsation in swelling decreased. Blood pressure 120. Author thought lesion was either a tortuosity of internal carotid just before it entered carotid canal or an aneurysm of the vessel. He inclined to latter view because of luetic history.

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Discussion.—Dr. Rudolph Matas (New Orleans, La.).—I always look forward to Doctor Winslow's papers with a keen anticipation of the profit and pleasure I expect to derive from them. For a number of years he has given us the benefit of his patient and painstaking bibliographic researches on the history and statistics of some of the rarer aneurysms of the carotid tract, of which his study of those of the extracranial carotid, in 1925, and of the temporal artery in last year's Transactions are notable examples. This year, in association with Doctor Shipley, he has brought to us a complement to his previous studies on the extracranial internal carotid which covers his experience during the intervening years and completes to date a monograph which will endure in the literature as the most complete record of the surgery of this relatively rare and particularly dangerous type of carotid aneurysm.

On previous occasions I have referred to the merit of Doctor Winslow's work, which is not that of the mere compiler of the statistics of others, but of the one who seeks at their sources the authentic facts upon which to base his conclusions. This is an ungrateful task because few appreciate the time and labor involved in these researches, except those who delve in the depths of the work and obtain pleasure from their findings. It is unfortunate, also, that the more differentiated a specialty is, the less likely it is to interest the general workers who are only concerned with the conclusions. Sometimes, those of us who labor in this special field, *i.e.,* vascular surgery, in our enthusiasm imagine that everyone is as much interested in our work as we are, only to discover that we are simply boring our colleagues with the details of our enterprises. None the less, though the problems of vascular surgery are highly specialized, there is no practicing surgeon who can avoid or afford to ignore them.

In connection with Doctors Winslow and Shipley's survey, I am glad to contribute a brief statistical synopsis of my personal experiences in the surgery of the internal carotid aneurysms which has not yet been published and which, I trust, may serve as an appendix to his record.

STATISTICAL SYNOPSIS OF DOCTOR MATAS' PERSONAL EXPERIENCES IN THE SURGERY OF INTERNAL CAROTID ANEURYSMS

1888–1936

Total carotid ligations for aneurysms..... 81
Five deaths; mortality 6.6 per cent
Total operations for aneurysms of the internal carotid tract, including 12 arteriovenous (pulsating exophthalmos)..... 18
One death; mortality 5.5 per cent
Total operations for aneurysms of the extracranial internal carotid..... 5
Ratio of extracranial to intracranial aneurysms (5 : 18) or 27 per cent extracranial
Ratio of extracranial to total (308) aneurysms treated by Doctor Matas, including 254 operated cases (5 : 308) or 1.6 per cent
Ligations or aluminum bands applied directly to the internal carotid..... 6
One death; mortality 16.6 per cent

The fatal case was that of F. S., colored male, age 42, who had a pathologic (luetic) aneurysm of the left common carotid at its bifurcation. A band was applied to the *internal* carotid, on its distal side, between the aneurysm and the skull, April 22, 1922. Cerebral symptoms developed 18 hours after the occlusion. The band was removed and circulation in the internal carotid restored. The patient, however, died, April 24, 1922, in coma, 48 hours after the occlusion. Autopsy revealed extensive thrombosis in left middle cerebral artery. The circle of Willis was markedly atheromatous and showed miliary dilatations. The artery at the seat of constriction by the band showed no pathologic change.

In addition to the six ligations of the internal carotid for extracranial lesions, there was one case (Miss I.) of internal carotid aneurysm treated by a band on the *common* carotid with perfect recovery.

In seven lesions of the internal carotid, four followed tonsillectomy, or 57.14 per cent caused by tonsillectomy or tonsillar infections.

In addition to the seven extracranial lesions of the internal carotid, there were 12 cases of pulsating exophthalmos, in six of which the common carotid was ligated, with no fatalities. Ligations for arteriovenous aneurysms of the carotid tract, including the internal, are relatively free from cerebral complications.

REMARKS.—(1) My experience, like Doctor Winslow's, confirms the classic belief that the ligation of the *internal* carotid is more liable to fatal cerebral disturbances than the ligation of the *common* carotid, as the former excludes the reversed (collateral) flow through the external carotid.

(2) Cerebral disturbances after the ligation of the internal carotid are essentially caused by cerebral ischemia which may be due to a number of factors, notably, thrombosis in the circle of Willis; but not embolism, as the normal flow in the artery is reversed after the ligation: *to* and not *from* the ligature.

DR. NATHAN WINSLOW (closing).—It is remarkable that these cases come to the general surgeon rather than to the nose and throat men. Of our four cases, three were operated upon by Doctor Shipley and one by myself. My patient was a Negress and a Negro doctor had attempted to plunge a lance into the supposed abscess; fortunately he did not succeed in his purpose, and the woman sought my advice.

FURTHER OBSERVATIONS ON THYROID DISEASE IN A NONENDEMIC AREA

AN ANALYSIS OF SIX HUNDRED SIXTY-TWO SURGICAL CASES AND SIXTEEN NONSURGICAL DEATHS

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FROM THE DEPARTMENT OF SURGERY OF THE SCHOOL OF MEDICINE OF LOUISIANA STATE UNIVERSITY AND THE RECORDS OF CHARITY HOSPITAL IN NEW ORLEANS

IN 1933,[1] and again in 1934,[2] we reported a series of 341 surgical cases of thyroid disease treated in Charity Hospital in New Orleans between January 1, 1927, and April 1, 1934. Herewith, we are adding to our first reports 321 surgical cases of thyroid disease treated in the same institution from the latter date to July 1, 1936 (Table I). This gives us a total of 662 cases of thyroid disease for a period of nine and one-half years in a region in which goiter is not only nonendemic but also relatively and absolutely infrequent. We make that statement deliberately, with full knowledge of the recent reports by the United States Public Health Service[3, 4] to the effect that in certain areas of Louisiana the incidence of goiter is as high as it is anywhere in the world. For that matter, surgeons of this community have long been aware of these isolated endemic areas. Their existence, however, does not alter the fact that even when they are given their full value, the incidence of thyroid disease in this part of the world is still very low. That is proved by the fact that it required a period of nine and one-half years to collect 662 surgical cases of this disease from an institution which, during that same period, had admissions of approximately 456,000, almost one-half of which were surgical.

TABLE I

INCIDENCE AND MORTALITY OF THYROID DISEASE IN CHARITY HOSPITAL IN NEW ORLEANS FROM JANUARY 1, 1927, TO JULY 1, 1936

First Series.....	341 cases	26 deaths	7.6 per cent
Second Series.....	321 cases	12 deaths	3.7 per cent
Combined Series.....	662 cases	38 deaths	5.7 per cent

Certain comments which we made in our previous communications ought to be repeated at this time. Now, as then, we must begin with an apology for reporting so small a series, in the face of the thousands of cases reported from true goiter centers elsewhere. But the very infrequency of thyroid disease in this community is both our explanation and our justification. Very few reports from the South have been made. Jones'[5] and Clifton's[6] series from Atlanta, Harris'[7] from Shreveport, Street's[8] from Vicksburg, about complete the list. Mahorner's[9] and Cohn's[10] cases from New Orleans are included in our own comprehensive series, which is, we believe, the largest number of cases so far put on record from a nonendemic area. All of these reports go

to prove the point we made in our previous communications, that any pathologic state is most a problem where it is least a problem. Furthermore, our achievements in thyroid disease furnish us no cause for pride, and they are not likely to be materially bettered until we bring the facts into the open and study them from the dual standpoint of what we have done and what we have left undone.

We pointed out in our first papers that as a consequence of the infrequency of thyroid disease in this community, no surgeon can have a very extensive experience with it. In the first series, the 341 operations were performed by 37 surgeons. In the second series, the 321 operations were performed by almost the same number, 38, though not always by the same surgeons. Combining the two series, we find that the 662 operations were performed by 54 surgeons, a state of affairs which clearly prevents any single man from operating upon a very large number of cases, and which forces all of us to gain our experience almost literally by the trial and error method. It follows, too, that with such a limited number of cases, it is almost impossible to train interns, anesthetists and nurses in the proper conception of thyroid disease or to make them realize the deadly potentialities of the toxic variety.

We emphasized another very serious consideration in our earlier papers, the practical impossibility, under the circumstances prevailing in Charity Hospital, of properly preparing hyperthyroid patients for operation. The complacent and entirely correct pronouncement of a recent writer, that a ward bed for an hyperthyroid patient is naturally out of the question, falls strangely upon the ears of those who are accustomed to include in their orders for such cases the underlined instruction: "Do not put anybody else in bed with this patient." All wards are overcrowded, the colored more so than the white—a serious consideration, in view of the higher mortality apparently inherent in thyroid disease in that race—and the absolute quiet which is the basic desideratum of preoperative preparation simply cannot be attained. We might add that the situation will be, if anything, worse while the new hospital is in process of construction. At least one case, not included in the series we are reporting, has already shown us the tragic consequences of the unavoidable noise and confusion incident to that project.

We noted in our former communications certain other facts: The chaotic nomenclature of thyroid disease, which all too frequently seemed to arise from an underlying confusion of thought concerning it; the unwise and inconsistent use of Lugol's solution; the tendency among both interns and staff to ascribe toxic manifestations to all varieties of thyroid disease, toxic and nontoxic alike; the abnormally high basal metabolic rates, which we attributed to the unpropitious circumstances under which these tests must be taken, circumstances which, we fear, are likely to be even worse in the immediate future than they were then, because of the cramped quarters and general confusion associated with the present hospital construction. These and other matters we discussed, and we pointed out the inevitable end-result, a mortality several

times higher than the mortality of thyroid surgery in endemic areas, even in the hands of the average surgeon, and very many times higher than the mortality of such surgery in the hands of an expert.

We begin our new discussion with the statement that, just as in the first series we reported, we have taken the liberty, in a fair number of cases, of reclassifying these patients according to a study of their records. That is by no means, of course, equivalent to a study of the patients themselves, but it still permits a detachment of outlook which seemed sadly lacking in many of the records we studied. Most important of our changes was the removal of patients from the toxic to the nontoxic group, an apparent impertinence in which we believe we were fully justified. We have commented before on the tendency of the historians to paint in all cases the picture of toxic disease, and the tendency of the staff to accept such a history without question for purposes of diagnosis and treatment. Lord Horder[11] has recently and properly called attention to the madness that seizes us all when the word thyrotoxicosis is bandied about, and it is quite evident that such a warning is needed, and should be heeded, in our surgical community. In the first place, the fact that a Negro has "pop eyes" does not constitute grounds for assuming, without further evidence, that he has exophthalmic goiter. Persons brought up in the South know, even though in these circumstances they seem to forget it, that protruberant eyes are frequently seen in Negroes, and a simple question as to the duration of the supposed exophthalmos would promptly eliminate the diagnosis, for it would show that the supposed pathologic state had been present almost since birth. In the second place, ignorant or neurotic patients, and particularly Negroes, are notoriously prone to exaggerate their "miseries" and to furnish any symptoms expected of them. The historian who begins to secure the anamnesis with the textbook picture of Graves' disease in his consciousness is very likely to emerge from the endeavor with the same picture on the chart. As for the basal metabolic rate, the time has long since passed when a single high reading, or even repeated high readings, are regarded as of independent value in diagnosis. In other words, it takes more than a combination of nervousness and an enlarged thyroid, even with the further evidence of a high basal metabolic rate, to justify the diagnosis of toxic thyroid disease. Let us quote a case in point:

Case Report.—A Negro woman, age 40, presented herself with the story of a growth in her neck which she had first noted ten years previously, immediately after her fourth pregnancy. It had remained stationary until four months before admission, since which time it had grown very rapidly. An unusually level headed intern noted on the chart that the patient had no symptoms of any sort, toxic, nervous, cardiac or mechanical; she simply desired the growth removed because its presence was disfiguring and because the recent rapid increase in size had frightened her. Her temperature, pulse and respiration were always within normal limits. Following subtotal thyroidectomy, without special preparation, she made an entirely uneventful recovery and was out of the hospital on the sixth postoperative day. Merely on the basis, so far as we could determine, of one basal metabolic rate reading of plus 35 and another of plus 23, a staff man was less wise than his intern and labeled the case as a toxic nodular goiter.

Such cases we have taken the liberty of reclassifying. We did not feel at liberty to drop from the series certain cases of supposed thyroid disease in which we doubted whether the symptoms complained of had anything to do with the thyroid. One such patient, for instance, operated upon two years ago with the diagnosis of diffuse toxic goiter, had as her chief complaint a constantly sore throat. She is in the hospital at the present time, not in the least benefited by her thyroidectomy, and still complaining of her sore throat, for which the nose and throat staff, who probably should have been consulted originally, have advised tonsillectomy. We reclassified such cases, even though we did not take the liberty of eliminating them.

Another small group of patients seem to have been submitted to surgery on the basis of "first catch your thyroid." They came into the clinic or the hospital for other conditions, usually recurrent appendicitis or gynecologic states, and their enlarged and symptomless thyroids were observed and promptly removed. The tendency was particularly notable and is particularly to be regretted, as we shall point out later, in young Negro girls. These young girls very frequently exhibit enlarged necks, and their goiters, if they really exist, we believe are better treated by other measures. In spite of careful study of the records and a serious endeavor at proper classification, we are still confronted with a certain proportion of cases which are either incorrectly classified as toxic, or which are really not thyroid disease at all. As a result, the inclusion of the latter group has altered for the better our general mortality for thyroid disease, while the inclusion of nontoxic cases in the toxic group has had the same effect on our mortality for the toxic disease. In short, and to speak quite bluntly, we believe that because of such errors our thyroid mortality is apparently lower than it actually is.

The results of our reclassification are shown in Table II. Why the death rate in Charity Hospital should reverse the usual situation elsewhere and be higher for the toxic diffuse than for the toxic nodular variety, we cannot say. The figures for the toxic variety are gratifying, in view of our comment in our first paper upon the marked increase in the incidence of this type of thyroid disease in the course of the period studied. It rose from ten cases, with no deaths, in 1927, to 31 cases with five deaths, in 1933. In the second period, even though, as we have just pointed out, we cannot accept our figures unreservedly, there is no doubt that both the incidence and the mortality have fallen. Even such uncompromising realists as ourselves must grant, too, that a general mortality of 3.7 per cent for the second period studied is decidedly better than a general mortality of 7.6 per cent for the first period studied, and we gladly admit that we can find valid explanations for at least part of the decrease.

First of all, the mortality fell because our preoperative preparation was far more intelligent in the second series than it was in the first. Even with our limited and utterly unsuitable facilities, we do try to keep our patients quiet, in beds by themselves, screened off from the rest of the ward, separated from the other patients and separated also from the well intentioned but ill advised

TABLE II

DISTRIBUTION OF THYROID DISEASE IN CHARITY HOSPITAL, NEW ORLEANS

Diffuse Nontoxic			
First Series	87 cases	6 deaths	8.0 per cent
Second Series	101 cases	1 death	0.99 per cent
Combined Series	188 cases	7 deaths	3.7 per cent
Diffuse Toxic			
First Series	94 cases	13 deaths	13.8 per cent
Second Series	61 cases	4 deaths	6.5 per cent
Combined Series	155 cases	17 deaths	10.9 per cent
Nodular Nontoxic			
First Series	77 cases	1 death	1.3 per cent
Second Series	99 cases	3 deaths	3.0 per cent
Combined Series	176 cases	4 deaths	2.3 per cent
Nodular Toxic			
First Series	83 cases	6 deaths	7.2 per cent
Second Series	60 cases	4 deaths	6.7 per cent
Combined Series	143 cases	10 deaths	7.0 per cent
Total Nontoxic Cases			
First Series	164 cases	7 deaths	4.2 per cent
Second Series	200 cases	4 deaths	2.0 per cent
Combined Series	364 cases	11 deaths	3.0 per cent
Total Toxic Cases			
First Series	177 cases	19 deaths	10.8 per cent
Second Series	121 cases	8 deaths	6.6 per cent
Combined Series	298 cases	27 deaths	9.1 per cent

attentions of their relatives and friends. The difficulty of isolating a friendly Negro, however ill he may be, can be appreciated only by those who have made the attempt. We feed these patients on high carbohydrate diets, a matter which the coöperation of the diet kitchen has completely simplified. We give them fluids freely, and we resort to preoperative infusions of glucose whenever there is any indication for such therapy.

One thing we are refraining from, which was responsible for a certain number of deaths in our early series, is the performance of miscellaneous, nonemergency procedures on toxic patients. The extraction of teeth before operation, for instance, undoubtedly was at least partially responsible for death in two of the four cases in which it was done, as were similar apparently trifling procedures in three other cases. Bayley,[12] in an excellent study of thyroid crisis, has pointed out the unwisdom of employing at certain times even such immediately applicable diagnostic measures as basal metabolic rate studies or roentgenologic examinations, and our experience, limited though it be, amply corroborates his statement.

We are using Lugol's solution more wisely than we once did. In the second series as in the first it was still occasionally used in the type of case in

which it is most positively not indicated, that is, the diffuse and the nontoxic nodular varieties, but such errors were increasingly infrequent. It was still used, as it was in the first series, for absurdly long or absurdly short periods of time, from one to 90 days, to make an honest confession. But as a rule the administration covered a period between nine and 15 days, a more or less reasonable limit in view of what histologic studies have shown concerning its effect on the thyroid tissue.

Lugol's solution is still, unfortunately, being used for long periods of time as a therapeutic measure, before the patient is turned over to the surgeon. That is not the surgeon's fault, of course, though it in no wise lessens his anxiety and responsibility when the patient finally reaches him, entirely refractory to iodine and deprived of its benefits in immediate preoperative therapy. Sometimes, of course, the circumstances are beyond anybody's control. When the patient takes iodine on the advice of her friends, or on the basis of her own perverted and imperfect knowledge, or out of a mail order catalogue—as actually happened twice in this combined series—certainly nobody in the medical profession can be held responsible.

Our chief complaint against the use of Lugol's solution in this second series is that almost never was it begun with the definite idea that operation would be performed within a certain period of time. Sometimes it was begun before the patient's consent to surgery was secured, and she refused operation for days or weeks or even months after she had been definitely prepared for it. More than once iodine was ordered as soon as the patient entered the ward, even before the differentiation into toxic or nontoxic goiter had been made or before the case had been worked up at all. Lahey's[13] insistence that the surgical risk be evaluated when the patient is first seen, before any diagnostic measures have been employed or any treatment instituted, is an admonition all of us might well take to heart. We do not sufficiently realize that all iodine remissions are temporary remissions and that the use of this drug is indicated only for the control of an actual or impending crisis or as part of the preparation for a definitely planned operation. Here, as elsewhere, conditions in thyroid surgery have been enormously improved by its use, but there is frequently a casual and almost frivolous use of this agent which entirely destroys its effect and which we cannot too strongly condemn.

Our anesthesia is far more intelligent than it was. Ethylene is now the anesthetic of choice. Ether is still occasionally used, but in this second series avertin was not used at all, nor was local. The latter anesthesia, we believe, is contraindicated in a clinic in which the experience with thyroid disease is not very large—if indeed it ever should be used. The lightest and safest and most controllable type of anesthesia, if we may so express it, is necessary in patients with thyroid disease, and particularly in Negro patients, who are likely to furnish many respiratory problems. Adequate exposure is frequently difficult and tedious, in women as well as in men, because of the highly developed musculature of the neck, due to their performance of heavy work and their habit of carrying baskets of clothes, vegetables, moss and other heavy

objects on their head. In the Negro, again, because of delay and neglect the glands are likely to be large and adherent, they are frequently substernal, and compression and distortion of the trachea lead to respiratory difficulties of the gravest sort, both at operation and later, and in all types of disease. Most of our deaths from respiratory collapse occurred in Negroes with large, nodular glands.

Our postoperative treatment is far wiser than it was. The liberal use of opiates as long as the need for them exists is now almost routine. Restlessness is not permitted to develop. Lugol's solution is very frequently given after operation, the idea on our own service being to spare the patient the strain of its abrupt withdrawal. We have tided several patients over threatened crises by this particular measure.

Our sheet anchor in postoperative treatment is the use of glucose infusions. We use them in all cases in which operation has caused any degree of shock or hemorrhage, regardless of the type of the disease. We use them, indeed, almost routinely on our own service, and rarely on any service is the former practice followed of giving fluid by vein only when the need for it arises. Deaths from crisis have therefore decreased, convalescence is smoother, and the patients are discharged within a shorter time. Of the value of this method of therapy there can be no possible doubt.

There were 12 stage operations in the second series against only eight in the first, both ligation and lobectomy being included in this term. Undoubtedly the divided operation was a very wise precaution in many cases. In many other cases, however, it seemed to us that it was done without justification, on patients who were not toxic or only mildly toxic, and for no apparent reason other than to prove the rather obvious thesis that less surgery is always safer than more surgery. Of course its use under such circumstances proves nothing at all, and the economic rights of both patient and hospital are lost sight of in this trivial employment of a really valuable measure. Even stage surgery carries a risk in bad cases; there were six fatalities in the 20 cases in which it was employed. This is particularly true of ligation, which terminated fatally in two of the five cases in which it was done. We are more and more inclined to agree with Pemberton, that if the patient does not respond to proper iodine therapy, he is not likely to be saved by ligation. More than one Negro male has proved that point to us.

Drainage, we might interpolate here, is still not a matter of standardized practice on any service. We ourselves approve of it. We believe that it has no appreciable effect on the scar if it is done at the angles of the incision, and we believe also that it is a definite protection against hemorrhage, in that it reveals it immediately, and against infection. We were interested to note, in support of our position, that perhaps three-quarters of all cases in which hematomata formed, or in which convalescence was delayed by infections or by serous discharges from the wound, were cases in which drainage had been omitted.

We think we may fairly say, too, that in this second series we have much

less reason to reproach ourselves for deaths from hemorrhage, from shock and from technical errors than we formerly had. Our technic may be still imperfect but it is considerably better than it was. Pneumonia is a less frequent and less fatal complication because of more careful preoperative observation, a wiser choice of anesthesia, and the postoperative use of the oxygen tent and of inhalations of CO_2 as may be indicated.

One final point may be mentioned, that in the second series there was almost routine examination of the vocal cords before operation. This is as much a protection for the surgeon as for the patient. Even more care than usual is demanded in operating upon a patient one of whose vocal cords is already damaged, and if such damage is evident after operation, it is well for the surgeon to know whether it is his responsibility or whether it already existed. The precaution was omitted in a recent case on our own service, and when unilateral paralysis of the cord was discovered after operation, the surgeon was not a happy man until he discovered, from an overlooked clinic sheet, that his carelessness had been provided for there and that a luetic infiltration and not his poor technic was responsible for the trouble.

Thyroid disease in the Negro is so special a problem that it deserves some extended comment. The once general belief that toxic goiter did not occur in that race has long since been exploded. L. H. Hermann,[14] in 1931, reported 40 cases from Lakeside Hospital, and advanced the opinion that the Negro is quite as susceptible to toxic thyroid disease as is the white man. We question the fairness of a comparison between the type of Negro he dealt with in the North and the type of Negro with whom we must deal in the South, but we concur in his opinion, and would go farther. We believe that toxic thyroid disease, when it occurs in the Negro in this community, tends to be a severer and graver condition than the same disease in white individuals.

In our first series of 341 cases (Table III) of thyroid disease, 148 cases, just over 43 per cent of the number, occurred in the Negro, and of these, 73, roughly half of the total, were of the toxic variety (Table IV). In the second series of 321 cases, 169, 52.6 per cent, occurred in the Negro, but of these only 50, less than a third, were of the toxic variety. We noted, as we have already pointed out, in the later years of our first study a marked increase of toxic thyroid disease in this community, and especially in the Negro. In the first three years of that study there had been only 14 such cases in the Negro, with no deaths, whereas, in the last four years there had been 59 cases with nine deaths. We speculated whether in the general financial straits in which this country had found itself since 1929, and in the increasing strain of modern life, the Negro might be losing his normal placidity. In the second series that increase seems to have been more or less checked. On the other hand, there is practically no difference in the mortality for the two series, 12.3 per cent in the first (73 cases with nine deaths) and 12 per cent in the second (50 cases with six deaths). That practically stationary mortality must be compared with the mortality in the white toxic group, which has fallen very decidedly: In the first series the mortality was 9.6 per

cent (104 cases with ten deaths), whereas, in the second it has fallen to 2.8 per cent (71 cases with two deaths).

TABLE III

RACIAL INCIDENCE AND MORTALITY OF THYROID DISEASE

First Series			
White	193 cases	11 deaths	5.7 per cent
Negro	148 cases	15 deaths	10.1 per cent
Second Series			
White	152 cases	3 deaths	2.0 per cent
Negro	169 cases	9 deaths	5.3 per cent

TABLE IV

RACIAL INCIDENCE AND MORTALITY OF TOXIC THYROID DISEASE

First Series			
White	104 cases	10 deaths	9.6 per cent
Negro	73 cases	9 deaths	12.3 per cent
Second Series			
White	71 cases	2 deaths	2.8 per cent
Negro	50 cases	6 deaths	12.0 per cent
Combined Series			
White	175 cases	12 deaths	6.9 per cent
Negro	123 cases	15 deaths	12.2 per cent

The general relationship between males and females in our first series was roughly one to 7.5 and the mortality for the whole male group was considerably higher than for the female group (Table V). Even more disturbing was the mortality for Negro males, 25 per cent. Those same tendencies are unfortunately continued in the second series of cases. The relationship is roughly one to eight, approximately the same as in the first group. The total male mortality is still several times higher than the total female mortality, and the Negro male mortality has risen to the appalling figure of 28.6 per cent. The risks of toxic thyroid disease in the male Negro furnish a very serious problem, which we shall consider again in our discussion of non-surgical deaths.

How to explain such discrepancies we do not know. It is usual for the male mortality of thyroid disease to be higher than the female, and the argument has frequently been advanced that there is a direct relationship between the infrequency of the disease in the male and its severity. But why should the general Negro mortality be so much higher than the white mortality (12.2 to 6.9 per cent)? And why should the disease be so highly fatal in the Negro male? It is our impression, though we cannot substantiate it statistically, that toxic thyroid disease is rather more frequent and more severe in the paler mulatto women, those with a large admixture of white blood, than in the more pure blooded types. But that does not hold true of

TABLE V

RACIAL AND SEX INCIDENCE AND MORTALITY OF TOXIC THYROID DISEASE

First Series			
White Male	20 cases	2 deaths	10.0 per cent
Negro Male	20 cases	5 deaths	25.0 per cent
White Female	173 cases	9 deaths	5.2 per cent
Negro Female	128 cases	10 deaths	7.8 per cent
Second Series			
White Male	19 cases	0 deaths	0.0 per cent
Negro Male	14 cases	4 deaths	28.6 per cent
White Female	133 cases	3 deaths	2.3 per cent
Negro Female	155 cases	5 deaths	3.2 per cent
Combined Series			
White Male	39 cases	2 deaths	5.1 per cent
Negro Male	34 cases	9 deaths	26.5 per cent
White Female	306 cases	12 deaths	3.9 per cent
Negro Female	283 cases	15 deaths	5.3 per cent
Total Male	73 cases	11 deaths	15.1 per cent
Total Female	598 cases	27 deaths	4.6 per cent

men. The most "impressively toxic" patients we have seen, to use Lahey's vivid phrase, were coal black Negroes. Their color does not necessarily eliminate the possibility of white blood, but there was certainly no visible evidence of it. When we are called upon to treat toxic thyroid disease in coal black Negro males, we can be certain that there are breakers ahead.

The high Negro mortality in thyroid disease can perhaps be partially explained by the general tendency of the race to postpone medical consultation in any disease in which pain is not an outstanding feature. In the nontoxic variety, this delay results in large, adherent, very nodular tumors. In the toxic varieties the chain of events is clear. The patient may have safely weathered a series of mild crises but their cumulative effects are seen in myocardial disease and extreme degrees of hypertension. Cardiovascular complications, quite aside from those of thyroid origin, are more frequent in the Negro than in the white patient, perhaps as the result of syphilis. It is a hospital rule that Wassermann examinations be made on all patients, and the surgeon who violates that rule on a Negro male ward is likely to regret his oversight. Syphilis is astonishingly frequent, and untreated syphilis and toxic thyroid disease do not go well together. It may be, too, that the same tendency prevails in thyroid disease as is evident in other so-called diseases of civilization, that when the native immunity to any pathologic process is lost, its manifestations are likely to be very severe. Our own studies have convinced us of the truth of this theory in peptic ulcer,[15] gallbladder,[16] and appendiceal disease,[17, 18] and carcinoma of the stomach,[19] liver[20] and gallbladder.[21] Whatever the reason, there is no doubt that when the Negro does pass into a thyroid crisis, his condition is likely to be very serious. We have personally

seen several cases in which, in spite of the proper therapeutic measures, the patients literally burned themselves up. And yet such crises, our personal experience shows, are actually less frequent and less severe among white patients in this community than they are elsewhere, where thyroid disease is endemic.

We are increasingly impressed with the new, rather generally accepted idea that the four varieties of thyroid disease, diffuse and diffuse toxic, nodular and nodular toxic—we prefer the nomenclature of the American Society for the Study of Goiter—represent not different types of thyroid disease but different degrees of the same basic pathology. On the other hand, we are thoroughly convinced that toxic thyroid disease, at least in this community, arises in a different way in the Negro and the white races. The disease in the white patient arises as virgin pathology, so to speak, in a gland which is not the seat of previous disease. In the Negro, just as is frequently the case in endemic areas, it arises on the basis of a previous nontoxic diffuse goiter. This type of goiter, which is first noted in adolescence, gives rise to no symptoms per se, and in the majority of cases no further pathologic change occurs in it. These Negro women are phlegmatic, they take life as it comes, their emotions are superficial and noisy rather than deep-rooted and essential, and there is no special strain upon the organism to fan the gland into activity. It is distinctly unusual, in our experience, to elicit from the Negro woman the history of any emotional strain or other event from which she can date the beginning of her thyroid symptoms.

Whatever be the reason, in the occasional case the inactive gland is fanned into activity and the diffuse toxic stage is reached. The white woman at this point is likely to present herself for treatment. The Negro woman ignores her disease, perhaps because her sensibilities are less keen and her toxic manifestations are therefore less marked. The gland, in its natural cycle, then passes over into the nontoxic stage and whether or not toxic manifestations again appear depends upon whether or not the factors, known or unknown, which were responsible for the original toxicity again become effective. In the occasional case it is quite possible that the gland may return to its original colloid or resting state, by the working of the law that all toxic thyroid disease will eventually burn itself out if it does not first burn out the patient. The fact that the Negro carries her thyroid enlargement longer than the white patient and endures her symptoms longer before she seeks medical aid is to us an explanation of the fact that nodular goiter is proportionately more frequent than the diffuse type in this race. Still further proof is the fact that colloid or diffuse goiter, the final stage of the cycle, was three times as frequent among Negro patients over 40 years of age as among white patients. Both these considerations lead to another point which should be emphasized, that the history of the disease is quite as important in diagnosis as are the immediate symptoms or the physical properties of the gland in one's hand.

The prevalence among young Negro women of enlarged necks, or enlarged thyroid glands, to speak more accurately, we have noted carefully since our

last communication. Then it was only an impression; now we know it to be a fact. Within the last six months we have had half a dozen such patients on our own service, whom we are observing at repeated intervals. We do not advise surgery for them, for the tendency to operate on such patients, as we have already said, we consider very unfortunate. We merely observe carefully the increase or decrease in the size of their necks in relation to the measures we have instituted, and we determine our further course by whether or not symptoms develop. Usually they do not. In the meantime, we cannot regard a symptomless enlargement of the thyroid gland as an indication for thyroidectomy.

In our first communication on this subject we called attention to Lahey's[22] statement that in his experience nonsurgical deaths from thyroid disease have been practically equivalent to postoperative surgical deaths. It was not the situation here, we stated, and it was not then. In the period covered by our first report there were only three medical deaths against 26 surgical deaths. But that situation has changed. During the period covered by our second study there were 13 nonsurgical toxic against 12 surgical toxic deaths. Even more disturbing, eight of those deaths occurred in 1935, in which year there were only two surgical deaths.

Those 16 deaths deserve very careful study. We may begin by saying that they were all toxic thyroid deaths. We discarded some six other fatal cases in which thyroid disease was present but in which it seemed to us to be the background rather than the principal cause of the fatality. Nine cases were of the diffuse and seven of the nodular toxic variety. All the nodular toxic cases occurred in Negroes, which substantiates our theory of the pathologic cycle of thyroid disease in this race. Six patients were white and ten Negroes. Twelve were females, and four, all Negroes, were male, proving again our point that toxic thyroid disease is a very deadly condition in the Negro male. In view of our continuous sunshine all year, and our constant supply of sea food and fresh fruits and vegetables, we are not usually inclined to put much stress on the seasonal incidence of goiter. On the other hand, there may be some significance in the fact—there would be elsewhere, we are sure—that 14 of the 16 deaths occurred between March and October, a period which in this latitude is always warm or very hot.

Most of these patients were admitted in a hopeless state, if not actually moribund. Five of them exhibited a psychosis, two were in coma, four were delirious, one had acidosis and one jaundice. In the patients from whom a history could be obtained (12) there was a story of a loss of weight varying from 40 to 64 pounds. Twelve had cardiac complications of some sort, usually very serious, and two had syphilis. All of the 12 whose histories could be secured had been treated for longer or shorter periods of time by medical measures. Four of them had taken Lugol's solution, and we have no doubt that if adequate questioning had been possible, the number would have been materially increased. The standard treatment for thyroid crisis was employed

in all cases, but these unfortunate individuals were doomed and nothing could be expected to help them.

Where the responsibility for these deaths lies is another matter. Most of the patients, as we have said, were admitted too ill for any treatment to be of avail—but surely some responsibility rests upon the internists who treated them medically for months and years without considering, as an alternative, the possibility of surgery. One of these patients died on our own service after transfer to us, while she was being prepared for operation. The preparation, we now realize, was hopelessly inadequate, but we did not comprehend until too late the full seriousness of her condition. We are in no position, therefore, to distribute blame. But these deaths do prove perfectly a point emphasized by Sloan,[23] that the mortality of this condition will never be reduced to the irreducible minimum until both physicians and surgeons realize that this is a constitutional and not a local state, a disease in which surgery is only one step in the cure, a disease in which constant cooperation between surgeons and internists, and not occasional formal consultations, forms the first principle of adequate therapy. These deaths certainly prove that it does not exist in this one.

Lahey[13] has recently said that with increasing experience he is more and more certain that deaths associated with hyperthyroidism are chiefly liver deaths, as is proved by the terminal jaundice sometimes present, the development of hyperpyrexia without infection and even before surgery, and the benefit achieved by the measures commonly used to combat liver damage, chiefly glucose infusions, forced fluids and transfusion. With that view we are in complete accord. Definite autopsy evidence of hepatic damage in thyroid disease has been furnished by a long list of observers and experimenters, including Weller,[24] Kerr[25] and Rusk,[26] Hashimoto,[27] Goodpasture,[28] Beaver and Pemberton,[29] and Cameron and Karunaratne,[30] to mention only a few. Our own studies[31, 32] on the so-called liver death have furnished additional evidence. The whole situation is well summed up by Mora,[33] in a recent comprehensive review of the liver lesion in thyroid disease. The interrelationship, he remarks, can be demonstrated clinically by the occurrence of jaundice, physiologically by increasing evidence of altered hepatic function, experimentally by evidence of hepatic dysfunction following the administration of thyroid extract and thyroxin, and morphologically by structural changes of varying degree. The practical application of these facts has been emphasized by Frazier and his associates, Brown, Frieman and North,[34, 35, 36] who have repeatedly pointed out the importance of intravenous glucose therapy as a prophylaxis against postoperative thyroid crisis and who have furnished convincing statistical proof of their claims. We are in full agreement with their conception that fatty changes in the liver are likely to be just as serious to the patient with hyperthyroid disease as is the simple depletion of the liver glycogen upon which heretofore we have tended to focus our entire attention.

In view of this new conception of thyroid disease, we have been impressed with the necessity of finding some method by which, if we may so express it,

the patient's state can be estimated in terms of the hepatic factor. Such a test would be of particular value in a nonendemic community such as this, where the individual surgeon's experience with thyroid disease is so limited that it is often very difficult for him to evaluate the patient correctly from the standpoint of surgical risk.

In the hippuric acid test of liver function, as devised by Quick[37] for use in hepatic and biliary disease, we believe we have found a method which fulfills these requirements better than any method hitherto available. It is a simple test, which can be easily used by any competent technician. It disturbs the patient as little as any test can. Except that it requires absolute accuracy —and what test should not?—the human equation is entirely eliminated, particularly the reaction of the patient, who always complicates the situation in such an estimate of operative risk as the basal metabolic rate, for instance.

To date, we have used this test in approximately 140 cases of various types of disease, the number including a series of control cases and also including 65 cases of various types of thyroid disease. We are naturally unwilling to go on record statistically until the number of completely studied cases is larger, but we are willing to make certain statements at this time. By means of this test we believe we can say with a fairly high degree of accuracy that one toxic patient is a good surgical risk and another, without very careful preparation, is a bad one. By repeated tests we believe that we can safely say, at the end of the planned preparation, that the patient has become a good risk, or a fair one, or remains a poor one, and must be submitted to operation with the full knowledge that the cards are stacked against a successful outcome. We are able to show by this test that the immediate result of operation, in toxic and nontoxic cases alike, is a prompt and sometimes very sharp decrease in the liver function, more marked, of course, in the former type of disease. We can prophesy that the patients who show a gradual rise of liver function after the first day or two will go on to prompt recovery, while patients who show a progressive decrease in liver function are likely to have a stormy convalescence and perhaps lose their lives. With further experience we hope to be able to determine the lower limit of safety in this test, so that we shall be able to say that some patients, no matter how urgently they may need thyroid surgery, had better be left to do their own dying, since the postoperative drop in liver function would be more than their badly damaged livers could endure.

An emphatic warning must be issued not to read into this test more than it can tell. It interprets liver damage in terms of liver function repeated tests interpret further alterations in the liver in terms of liver function, but a single test and repeated tests indicate nothing more. Most emphatically, this test does not indicate the type of risk presented by the so-called thyrocardiac patients, in whom the heart condition has probably become more important now than the thyroid state. It is most important to realize that in thyroid disease there eventually comes a time when the visceral damage wrought by the hyperthyroid state actually overshadows the initial hyperthyroidism. That

type of risk this test does not indicate, nor does it indicate the possible risk from respiratory failure and similar postoperative complications. Two recent cases have brought that lesson home to us very clearly. Both patients were Negroes, both exceedingly toxic on admission, both very carefully prepared for operation, both with liver functions, which had been not more than 40 to 50 per cent of normal on admission, restored practically to normal before operation. One patient, a male, died of congestive heart failure 36 hours after his second lobectomy, the other, a woman, of some type of respiratory failure, which could not be determined, three hours after an apparently normal reaction from operation. In neither case was there the smallest evidence of thyroid crisis. The evidence of heart failure in the first case was unmistakable, and the cardiac risk had been perfectly comprehended before operation was attempted. The second patient, with a normal temperature, blood pressure, pulse and respiration, simply stopped breathing. No liver function test or any other sort of test will prevent fatalities of this sort. We do believe, however, that if the hippuric acid test is used with a proper conception of its scope and limitations, it will be of material value in reducing the mortality of toxic thyroid disease so far as that mortality arises from hepatic damage.

SUMMARY

(1) To the 341 surgical cases of thyroid disease which we have previously reported from Charity Hospital in New Orleans, we are adding 321 similar cases, the combined series of 662 cases covering a period of nine and one-half years.

(2) The disease is a peculiar problem in this area. Because it is nonendemic, both general and individual experience with it is limited, and this comparative inexperience results, in combination with other factors, in a mortality several times higher than is reported from various goiter centers.

(3) The reclassification of the records was necessary in many instances, chiefly because of the tendency to consider nontoxic thyroid disease as toxic.

(4) The increasing incidence of toxic thyroid disease noted in our first communications has apparently been halted, and the mortality in the second series is less than half of the mortality in the first series. This decrease we attribute to better preoperative preparation, abstinence from nonemergency preliminary procedures, a wiser use of Lugol's solution, a more intelligent selection of anesthesia, and better postoperative treatment. Particularly important is the frequent use of glucose preoperatively and almost routinely postoperatively.

(5) Thyroid disease presents a very serious problem in the Negro, and particularly in the Negro male, in whom it is highly fatal, though no adequate explanation can be advanced.

(6) The disease arises on a different basis and runs a different course in Negro and in white subjects.

(7) Sixteen nonsurgical thyroid deaths are analyzed, ten of which occurred in Negro patients.

(8) The relationship of the thyroid gland and the liver is briefly discussed, and the hippuric acid test is suggested as an index of liver damage. Its scope and limitations are outlined.

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AN ANALYSIS OF THREE HUNDRED CONSECUTIVE THYROIDECTOMIES

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THE distribution of goiter in Alabama is largely confined to the northern part of the state, among the foothills of the Appalachian Mountains. Over 90 per cent of the cases reported here lived in this region. They were seen and treated in the eight years, 1928 to 1935, inclusive, and, living in a compact area, it has been comparatively easy to follow them. We have heard from every case either directly or indirectly, and in the great majority, we have seen them personally from time to time. The malignant goiters seen were beyond surgical help, and we undertook no thyroid operations for cardiac disease, because we were ignorant of the end-results.

The diagnosis of hyperthyroidism in mild cases is often quite difficult. There were 203 toxic cases and 97 nontoxic, making a ratio of practically two to one.

The border line between the slightly toxic and other conditions, particularly certain nervous conditions with a rapid heart, is often hazy and indefinite. In these cases, we have taken the stand that it is better to keep the patient under observation for months, and let time help in the diagnosis. In this way, there is less chance of imposing an unnecessary operation upon patients of the neurocirculatory asthenic type.

All cases with nodular goiters had roentgenologic examinations to determine the possibility of substernal adenomata.

As a general rule, cases with a pulse rate of 80, or under, were considered to be definitely nontoxic.

There was elevation of the blood pressure in 70 cases, and it was but little affected by operation. The typical high pulse pressure was often present in the younger toxic cases. I know of no other condition in which the systolic pressure is regularly almost twice the diastolic.

Age: The oldest was 73 years, the youngest six. The average age was 34.6 years. There were 141 patients 35 years of age or older; 159 were under 35.

Sex: There were 256 or 85.3 per cent females. There were 44 or 14.7 per cent males.

Race: There were 273 whites and 27 Negroes; 229 white females, 44 white males. There were no males among the Negroes. The Negro population of Alabama is about 39 per cent of the whole.

Types.—Toxic: 203 or 67.6 per cent were toxic. Of these, 133 were diffuse, and 70 were nodular.

Nontoxic: 97 or 32.4 per cent, nodular 83, diffuse (colloid) seven, fetal

adenomata two, aberrant thyroid two, chronic thyroiditis two, acute suppurative thyroiditis two.

Associated Conditions: One had a four plus Wassermann, three cyst of ovary, three diabetes, three pregnancy under four months, five severe myocarditis, three fibromyoma uteri.

Primary Symptoms in Toxic Cases in Order of Severity and Frequency. Nervousness 147, weakness 135, palpitation 91, loss of weight 67, exophthalmos 59, choking sensation 12, fever unexplained one. The greatest loss of weight was 121 pounds (from 211 to 90). This occurred during 12 months, during which time the patient took 30 drops of Lugol's solution daily. After operation in two stages, her weight returned in six months to 200 pounds. This case illustrates the futility of treating toxic goiter with iodine.

Pathology.—The pathology of goiter is somewhat confused. It is cyclic in nature, and different phases may appear at the same time in the same gland.

Diffuse Toxic: Here the gland is nearly always enlarged, is symmetrical and feels quite firm. This firmness is accentuated by iodinization. Microscopically, there is great hyperplasia of the epithelium, which is high and columnar, with infolding into the acini. The increasing epithelium largely replaces the colloid material, which may almost entirely disappear. This affords the anatomic basis for hypersecretion.

Nodular Toxic: Commonly called toxic adenoma. The most striking characteristic is its encapsulation. They vary in size from a match head to an orange, and are prone to undergo degeneration. Cystic formation is common. With each successive cycle, the cyst may enlarge, due to coalescing of adjacent cystic acini. Hemorrhage into cysts is frequent. Thyroid tissue surrounding the adenoma is hyperplastic and probably furnishes most of the thyroxin secreted.

Diffuse Nontoxic: The acini are large, filled with dense colloid material; the epithelium is low and flattened, and blood vessels are few. Scattering small areas of lymphoid hyperplasia can usually be found.

Nodular Nontoxic: The gross picture is essentially the same as that in toxic adenoma. Microscopically, the surrounding thyroid tissue does not show epithelial hyperplasia. Its chief offense is the causing of pressure symptoms, and it may become malignant. Ninety-six per cent of all malignant goiters arise in solitary adenomata.

Chronic Thyroiditis: Strangulation, destruction, and replacement of glandular epithelium by fibrous tissue; hypothyroidism often results. The gland is very hard, but is distinguished from carcinoma by the preservation of the normal lobulation and normal outline of the gland, although the latter may be indistinct.

Preoperative Preparation for Toxic Cases.—Rest in bed 22 hours a day in severe cases; up an hour morning and evening. Increased fluids by mouth or intravenously. High caloric diet. Lugol's solution of iodine by mouth, usually 30 drops daily. It can be given intravenously in glucose solution. In

patients who show but little remission with iodine, drastic removal of thyroid tissue is indicated.

Cardiac conditions, when present, were treated by the internist. Digitalis was given as in nongoiterous cardiac conditions.

When sufficient benefit was obtained from some, or all the above factors, operation was performed. Patients who were iodine-fast, or had been taking iodine for a long time, were taken off iodine for eight to ten weeks, and then reiodinized intensively for eight to ten days prior to operation.

There were 16 multiple stage operations; 14 were performed in two stages, while two cases were performed in three stages, ligation representing the first stage. At the first operation we removed the isthmus and one lobe, usually the right; six to eight weeks later, the operation was completed. The decision to perform the operation in two stages was definitely arrived at before operation, after carefully considering the general condition of the patient. It was not made during the operation.

Indications for Operation.—To prevent further damage to the heart and circulatory system, to arrest process of autodestruction, to relieve pressure symptoms, for cosmetic reasons, and in the adenomata cases to forestall toxicity, and to prevent the development of malignancy in discrete adenomata.

Operative Technic.—First and most important of all is a good exposure of the operative field. One should never hesitate to sever ribbon muscles. It is better to cut them than to risk injuring a nerve.

Removal of Adequate Amount of Thyroid Tissue: Only actual experience can teach this.

Ligation of Superior Poles: This materially cuts down bleeding and helps mobilize the gland.

Ligation of Middle Thyroid Vein: This should be accomplished early in the operation; otherwise, it is easily torn, retracts and causes annoying hemorrhage.

Removal of Isthmus from the anterolateral aspect of the trachea, thus removing pressure on it.

Ligation of Inferior Thyroid Artery is occasionally done, in order not to have to grasp later for bleeding points in the region of the recurrent nerve.

Removal of Pyramidal Lobe, when present.

Thyroid Remnant turned inward and sutured to the trachea, which helps to stop oozing, restores the normal outline of the neck, and in cases of secondary operation greatly facilitates the exposure of the gland.

Complete Hemostasis.

Closure Without Drainage.

Complications.—Drainage in thyroid surgery should be considered as a complication. It was deemed necessary in 25 operations, 15 of which were for substernal goiter. The drain was usually removed after one or two days.

Multiple Stage Operations were performed on 16 very severe cases, where it was feared that the complete operation might result in a fatality. As evidence of the severity of this group, two of them died within four months after

the operation; however, not from thyroid disease. One of them had gained 22 pounds and the other about 15.

Substernal goiter was present in 15 cases and offered no unusual difficulty in operation. They were all drained. Severe postoperative crises occurred in three cases and required energetic treatment. Lugol's solution may be given intravenously in 5 per cent glucose solution, as much as 50 minims in a liter, every three or four hours in the severe cases.

The oxygen tent was considered very helpful in two of these cases. Postoperative fibrillation occurred in seven cases and was definitely helped by giving quinidine. Postoperative bleeding was present in one case and was found to be in the skin flap where it was easily controlled. The trachea was accidentally opened once, where an adenoma had greatly compressed it. By suturing the muscle against the trachea, it caused no trouble, and this patient went on to develop a pyloric obstruction 18 months later, which was relieved by operation. There was one postoperative infection, from which there was no untoward result. Injury to the recurrent nerve occurred once in a patient, while removing a large adenoma, who had been operated on previously elsewhere. Tetany did not occur in any case.

The average stay in the hospital was 8.2 days and in the last 100 cases the average stay was only five days.

Anesthesia: Preliminary sedation was employed with good effect in all cases. We now give nembutal Gr. 3 one and one-half hours before operation and morphine Gr. 1/6, scopolamine Gr. 1/200, 45 minutes before operation. This is varied at times. In the weak and feeble, atropine may be substituted for scopolamine.

In 20 cases operated upon at the Charity Hospital, ether was used. In the early cases of this series, novocain infiltration was used in 48 private cases. At that time we did not have good anesthesia. Since 1930, with good anesthesia, we have operated upon the last 232 cases of this series with nitrous oxide and oxygen. Since September, 1936, we have used cyclopropane exclusively, and like it much better than any other anesthetic for thyroid surgery.

Pulse Rate: The average pulse rate on admission was 100. The average pulse rate in a group of 116 very toxic cases on admission was 110. The average postoperative pulse rate was 80.

Basal Metabolism: This was ascertained in 282 of the 300 cases. The highest obtained was plus 78. This same case had been estimated under the same conditions elsewhere at plus 104. The lowest basal metabolism in a definitely toxic case was minus five. The lowest basal obtained on admission was minus 21. A good many of the nontoxic cases, particularly the nontoxic nodular type, who had a low basal metabolic rate, approximating minus 15, showed a rise to normal after the operation.

Results: Those obtained on the whole were highly satisfactory. There was complete operative recovery of 299 cases. One patient died four days after operation. This patient was 67 years old, was unable to walk when she entered the hospital, had severe myocarditis, arrhythmia, edema, and should

not have been subjected to operation. She had been twice in the hospital before, and had been given roentgen therapy by her attending physician. Of the three recurrent cases, two have been reoperated upon, one of which still remains uncured. Five of the six persistent cases have been reoperated upon and four appear cured, while one continues to show mild hyperthyroidism. One hundred twenty-two, or 60 per cent, of the toxic cases gained an average of 15 pounds; 282 cases, or 94 per cent, of all types, either gained or remained stationary; 18, or 6 per cent, lost weight, and did not do well on the whole. The recurrent and persistent cases were among these.

One of the striking results of operation was relief from nervousness. In only one case was there a marked relief from hypertension. This was in a boy of 19, and this did not become manifest until a year had elapsed.

The follow up record may be of interest: One young woman, pregnant, very toxic, developed mania; was incarcerated; recovered. One young man developed perforated duodenal ulcer eight months after thyroidectomy. A pyloroplasty was performed and he recovered. One woman, age 52, developed pyloric obstruction 18 months later; a gastro-enterostomy was performed, and she has remained well four years. One young woman developed hypertrophy of the pyramidal lobe, which was disfiguring, 12 months after operation. It was removed. A man, age 34, very toxic, died eight weeks after hemithyroidectomy from acute lobar pneumonia. He was sick only three days. Up to the time of his last illness, he had gained 15 pounds and was ready for the second stage operation. One woman, age 36, died four months after the second stage operation from complications following influenza. She developed a thrombosis of the right axillary vein with great swelling and edema of the extremity. She had gained 22 pounds between the operation and the beginning of her last illness. One young woman died 12 months after operation in diabetic coma. One woman, age 50, died, in a neighboring state, following an abdominal operation, in which the gallbladder and an ovarian cyst were removed at the same time. Two patients more than 65 years of age died five years after operation, with symptoms of hypertension and cardiac failure.

In conclusion, we wish to say that nothing original is claimed in presenting this small series of cases. Perhaps the only worth while point is that a low mortality rate was obtained. This we feel was the result of attention to details, and, particularly, by refusing to operate upon patients not properly prepared.

CONCLUSIONS

(1) Hyperthyroidism is at times difficult to diagnose.

(2) Great care should be used in arriving at the decision to operate upon bad risk cases.

(3) Multiple stage operations are indicated in some of the most severe cases.

(4) Correct and complete preoperative preparation is essential to the patient's safety, and will reward the surgeon with a gratifying mortality rate.

TOTAL THYROIDECTOMY FOR HEART DISEASE

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THE proper evaluation of any new therapeutic agent or method is always difficult—particularly so when the disease being treated is chronic in character and subject therefore to natural exacerbations and remissions. If the operation of total thyroidectomy proves of unquestioned value in the treatment of congestive heart failure and angina pectoris, much will be added to the armamentarium of those who labor, often with so little success, to relieve the chronic cardiac invalid. It is, therefore, of importance that repeated attempts be made to evaluate this method of treatment. We believe that a sufficient number of patients have now been treated by total thyroidectomy, and that enough time has elapsed since the operation was first advocated, in 1933, by Blumgart, Levine and Berlin to clarify our ideas in regard to the results. That sharp differences of opinion as to the value of this procedure do exist may be readily ascertained by even a casual survey of the literature pertaining to the subject. Clute[9] is as yet not convinced that the results obtained warrant the operation except in cases of severe angina. Mont Reid[47] thinks the entire procedure of total thyroidectomy unsound and unjustified. Trout[56] has not been sufficiently impressed with the duration of improvement to advise any patient to subject himself to the hazards of the operation, and Moore[35] feels it so unphysiologic that he has never advised its performance. On the contrary, Pemberton[41] thinks the operation rests upon sound physiologic principles, and Brenizer[5] has been quite enthusiastic as to the results he has obtained. Between the vigorous endorsement of the originators and other proponents of the operation and the strongly adverse opinions of surgeons and cardiologists of equal repute, there must be a middle ground far more tenable, and it is quite likely that somewhere between the extreme views mentioned there lies the real truth.

To rely entirely upon the published data concerning any new therapeutic procedure is apt to lead one to erroneous conclusions. It is uniformly true that advocates of any new method are more prolific in their writing than are those whose experience has been less fortunate. We propose, therefore, to tabulate, in so far as we are able, data concerning all cases on whom the operation has been performed, even though many of these cases have not appeared in the literature, and, from this study, to surmise if the end-results obtained show the operation to have merit; to indicate what patients, if any, should be subjected to the operation and to make certain general comments pertinent to the entire procedure. Our own cases we present with the general

tabulation. The material offered was obtained from a fairly complete survey of the literature and from an inquiry sent to all members of the American, the Southern and the Western Surgical Associations, the American Association for the Study of Goiter and certain other American clinics. Six hundred seventy-seven inquiries were mailed and answers were obtained in 342 instances. Only 59 individuals or clinics reported a definite number of cases in which the operation had been performed. A number of other individuals appeared to have had some experience with the operation, but the data given were not in form adaptable for our studies.

We have data concerning 362 upon whom the operation was performed. Except in regard to complications, the results will be recorded separately in the angina pectoris and the congestive heart failure groups. Information concerning complications was obtained on 291 cases. In this group tetany occurred 30 times (10.3 per cent), though fortunately, in all but one instance, it was transient in duration; in this instance it appears to have been the cause of death. Injury to the recurrent laryngeal nerve occurred on 24 (8.2 per cent) occasions, and this complication likewise was usually of transient nature. In no instance was there bilateral nerve damage. As would be expected, all operators have been alert to these major complications, which, at least theoretically if not always practically, are avoidable. We believe that serious complications are not sufficiently frequent to militate greatly against the procedure. It is, however, probably true that total thyroidectomy has been attempted chiefly by those possessing a relatively wide experience with thyroid surgery and, unquestionably, the operation ought not to be attempted by others.

We do not wish at this time to discuss the rationale of total thyroidectomy in the treatment of heart disease. Probably multiple factors are involved. Of the various views expressed, it appears to us that the one most tenable would indicate that we should expect the best results in patients with angina pectoris. For that reason we have elected to consider the results in angina and in congestive heart failure separately.

For congestive heart failure there have been 229 operations performed. In this group there occurred 24 (10.48 per cent) operative deaths. We have considered as operative deaths those individuals who died within a short time after operation, usually within the first week. This is considered representative of the amount of immediate increased hazard which the operation entails.

The results are grouped in four classes. By "excellent," we mean that there has been no recurrence of symptoms or signs, in spite of activity greater than that enjoyed preoperatively. The majority of these individuals have been restored to a more normal life. "Moderately improved" indicates that the attacks are less frequent and less severe, even with increased activity. "Slightly improved" indicates that attacks are somewhat less frequent and less severe and the patient more comfortable though the range of activity is little changed. All other results are classified as showing "no improvement." The results given here do not include the operative deaths. To do so would naturally increase the percentage of failures, but it must be remembered that

a considerable number of the cases here reported were among the first operated upon at the Peter Bent Brigham Hospital and the Beth Israel Hospital before either of these institutions had thoroughly established their criteria for the selection of cases, and many of those who represent failures are now known to have been poor subjects for the operation. Each of these institutions, pioneers in this field of work, is now performing a lesser number of operations on a more carefully chosen group of patients, and their results will be found correspondingly improved.

The immediate operative mortality encountered in the series being reported of patients suffering congestive heart failure compares very satisfactorily with that of 12 per cent reported by Mixter, Blumgart and Berlin[33] in an experience of 46 cases, and is only slightly higher than that of 9.3 per cent found by McCreery and Weeks[34] on 150 cases reported from their questionnaire.

In our series of collected cases, 71 patients, or 34.63 per cent, showed excellent results. Fifty-nine, or 28.78 per cent, showed moderate improvement, six, or 2.92 per cent, were only slightly improved and 69, or 33.65 per cent, were classed as unimproved and failures. Our data correspond reasonably well with those of Weeks and McCreery, though Mixter, Blumgart and Berlin's figure of only 7 per cent failure is far lower. If we combine groups one and two, considering the total as constituting those with satisfactory improvement, a percentage of 63.41 per cent is obtained in comparison with 81.1 per cent reported by Weeks and McCreery and 76 per cent by Mixter, Blumgart and Berlin. On the contrary, Clark, Means and Sprague[8] found only one-fourth, or 25 per cent, of their cases to have shown improvement, and three-fourths, or 75 per cent, were recorded as failures. We believe that the true figure for satisfactory results, following thyroidectomy for congestive heart failure, to be approximately 60 per cent.

We desire now to consider the data obtained from a study of 133 operations performed for angina pectoris. In this group, the operative mortality was 3.75 per cent (five), essentially the same as recorded in Weeks and McCreery's series (3 per cent). In the smaller group of Mixter, Blumgart and Berlin, there were no deaths. In the series being recorded, 71 patients, or 55.46 per cent, showed excellent results. Thirty-six, or 28.12 per cent, were moderately improved. Five, or 3.9 per cent, were slightly improved and 16, or 12.5 per cent, were classed as failures. These figures correspond fairly closely with the group of Mixter, Blumgart and Berlin and of McCreery and Weeks. Again considering the first two classes as satisfactory, we find 83.58 per cent good results, as compared with 85 per cent and 91.4 per cent, respectively, by the two observers previously referred to. We believe that the correct figure for good or satisfactory results in the cases of angina pectoris lies between 80 and 90 per cent.

Our statistics, tabulated in Table I, indicate that neither the operative mortality nor the type and number of complications to be expected are sufficiently great to deter us from applying this operation *when indicated*. It appears that there is a slightly more than 50 per cent chance of satisfactory

TABLE I

STATISTICAL RÉSUMÉ OF 226 CASES OF CONGESTIVE HEART FAILURE AND 133 CASES OF ANGINA PECTORIS OPERATED UPON BY TOTAL THYROIDECTOMY, AND THE RESULTS OBTAINED*

Surgeon	Cardiac Condition		Results							
	C. H. F.	Angina	C. H. F.				Angina			
	Number of Cases		No. 1	No. 2	No. 3	No. 4	No. 1	No. 2	No. 3	No. 4*
Allen, Duff[1]	9	2	2	2		5	1			1
Beth Israel Hospital[2]	46	23	25	12		3	7	12		4
Bisgard, J. O.[3]	2	2		1		1	2			
Booth, A. E.[4]	2	0		2						
Brenizer, A. G.[5]	3	5	1	1			5			
Brooks, Barney[6]	1	0				1				
Chapman, D. G.[7]	1	0	1							
Clark, R. J.[8]	19	2		3		13		1		1
Davidson, T. C.[10]	2	1	2				1			
Duke University[11]	1	0	1							
Eutine, Donald[12]	2	0		1						
Fahrni, G. S.[13]	0	2					2			
Felger[14]	3	4	1	1		1	3			1
Gallie, W. E.[15]	4	5	1			2	5			
Gatewood[16]	1	1	1				1			
Gillett, N. W.[17]	3	2	1	1				2		
Graham, Roscoe[18]	0	4					3			1
Grove, Lon.[19]	2	0	1		1					
Jackson, Arnold[20]	5	1	1	2		1		1		
Jones, Elmer[21]	0	1						1		
Lee, W. E.[22]	2	0				2				
Lehman, E. P.[23]	1	0				1				
Link, G.[24]	1	0	1							
Maes, Urban[25]	6	0		2		2				
Mahon, G. O.[26]	1	1		1				1		
Mastin, E. V.[27]	3	0	2	1						
McClure, R. D.[28]	2	3	1	1			1	2		
McGhee, J. L.[29]	2	0	1			1				
McNealy, R. W.[30]	3	1		1				1		
Miller, E. L.[31]	4	3	3	1			3			
Miller, Richard H.[32]	8	0		1		6				
Miscellaneous	5	0		3		2				
Morton, Bruce[36]	3	0				3				
Morton, J. J.[37]	6	2				6	2			
Ochsner, Alton[38]	4	1	1	2		1	1			
O'Keefe, M. E.[39]	4	0		3		1				
Parsons, Barclay[40]	5	5	1	3		1	1	2		1
Parsons, W. H. and Purks, W. K.	2	3	1	1			1	1		
Percy, N. M.[42]	3	1	1	1		1	1			
Peter Bent Brigham Hospital[13]	15	27	6	2	3	3	12	8	4	
Phemister, D. B.[44]	1	0				1				

* No. 1, Excellent results. No. 2, Moderate improvement. No. 3, Slight improvement. No. 4, Failure to improve.

TABLE I (*Continued*)

Surgeon	Cardiac Condition		Results							
	C. H. F.	Angina	C. H. F.				Angina			
	Number of Cases		No. 1	No. 2	No. 3	No. 4	No. 1	No. 2	No. 3	No.* 4
Ravdin, I. S.[45]	9	12	3			6	7			5
Reichert, F. L.[46]	1	1		1			1			
Rice, Carl O.[48]	9	0	5	4						
Schwyzer, A.[49]	1	0			1					
Schier, H. V. B.[50]	0	5					4			1
Smith, M. K.[51]	0	1								1
Soley, Mayo[52]	0	1					1			
Straus, D. C.[53]	2	2				2		2		
Sturgeon, C. T.[54]	0	2					1	1		
			1	2	3	4	1	2	3	4
Taylor, W. A.[55]	0	1					1			
Weeks-McCreery[34, 57]	10	6	3	4		2	4	1	1	
Wise, W. D.[58]	5	0	4	1						
Yates, J. L.[59]	2	0			1	1				
Total	226	133	71	59	6	69	71	36	5	16

improvement in cases with congestive heart failure and somewhat better than 75 per cent satisfactory results in angina pectoris. Quite candidly it must be said that we have been surprised at the extremely low operative mortality in the angina pectoris group and at the extremely high percentage of satisfactory improvement in this group.

Aside from statistical data concerning the results of total thyroidectomy in heart disease, there are other considerations of importance. No one may say as yet, definitely, whether the operation prolongs life. It is admittedly only a form of symptomatic treatment which in no way alters the underlying cardiac pathology. We must bear in mind that we are treating one disease by the substitution of another, even though the latter, myxedema, is milder and more amenable to treatment. One also must insist that patients operated upon remain under treatment for the remainder of their lives. The internist who recommends the operation or the surgeon who executes it must bear in mind that if the patient, by reason of one cause or another, falls into the hands of other physicians not willing or able to continue the proper post-operative care, he becomes a pitiable problem. On the contrary, it must in fairness be recalled that patients subjected to the operation have, in most instances, been totally incapacitated and have suffered miserably because of their cardiac disease which has failed to respond to medical treatment; for surgery should not be resorted to under other circumstances. We are not justified in demanding perfection of a method applied to such patients.

Finally, we feel that total thyroidectomy is a valuable addition to the treatment of angina pectoris and congestive heart failure. It is imperative, however, that the accessibility of this procedure should in no wise lessen our attempts to control all cardiac problems by nonsurgical means. The selection

of cases must be very carefully considered. The greater probability of worth while results is to be expected in patients with angina pectoris.

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DISCUSSION OF THE PAPERS OF DOCTORS MAES, DRENNEN, AND PARSONS

DR. ADDISON G. BRENIZER (Charlotte, N. C.).—The explanation of the death in the Negroes was that the Negro with a thyrotoxemia of a grave type does not appreciate his condition and is usually a neglected case. Apparently Doctor Maes has encountered some very badly burned out cases in this race.

As to Doctor Drennen's paper with the small percentage of Negro incidence, I do not understand that, for there is no doubt that Negroes do have exophthalmic goiters. I think Doctor Bram of Philadelphia said he had never seen a Negro with exophthalmic goiter. The figures are very interesting to me. We do not like to quote our mortalities, for if the rate is good we might be suspected of having it too good, but I cannot understand how one surgeon in Birmingham has about one-third of 1 per cent and another, equally good surgeon, in New Orleans would have such a high mortality. My first 100 cases had a much higher mortality than my later ones, but I do not think that accounts for this discrepancy in mortality of these two authors. One surgeon may have a run of a certain type of severe cases of goiter or select such cases which yield a high mortality. This spring, three of my cases died in the hospital while being prepared for operation. It is obvious that they would all have died, had they been operated upon.

The distribution of the cases cited by Doctor Maes was among about 25 young surgeons, whereas the work of Doctor Drennen was by one man, who apparently has operated upon a class of case of less severity than those encountered by Doctor Maes. Doctor Parsons has quoted me in favor of total thyroidectomy, particularly in cases of badly damaged hearts with angina, where the patient should be markedly slowed down. While I am heartily in favor of total thyroidectomy in such cases, carefully selected, I have performed many less operations upon them than he has.

DR. WILLIAM D. HAGGARD (Nashville, Tenn.).—I think the differences between the New Orleans and the Birmingham statistics are probably overlooked by Doctor Brenizer because Doctor Maes reported a larger percentage of Negroes. I think Doctor Maes is to be congratulated upon the reduction of his mortality, as is Doctor Drennen for his excellent presentation of his experiences and results. I think Doctor Maes can be comforted by the fact that the Royal Infirmary of Edinburgh reported 12.4 per cent and the Royal Infirmary of Liverpool 12.8 per cent mortality. It would seem as if royalty was on the decline. In our last 300 cases we had only two deaths. I speak of this because I feel that we could have prevented these. One patient, an elderly, enormous woman, was operated upon successfully, but on the ninth day was found dead in bed. The other case was that of a tense, turgid man with acute hyperthyroidism that we did not sufficiently prepare, although he was given Lugol's solution, and did not use the best method for his particular case.

I think we must always take into consideration the mental attitude of the patient, for we know the patient who is nervous, tense and irritable is not a good risk. This man's basal metabolism went down to about 21, his pulse was 110, and we thought he was ready for operation. I belong to the school that believes in multiple operations, but in this case I thought I was so good I did not have to do that. I should have used only preliminary ligation. Personally, I would rather ligate and find out what they will stand. We have seen patients die even from ligation, but I think this man would have been saved by it. Our improvement has been so great with the use of Lugol's solution that we have lost sight of the necessity for choosing the multiple operation.

DR. FRANK H. LAHEY (Boston, Mass.).—I have been very much interested in these papers, and particularly to see the improvement that has taken place in New Orleans since Doctor Maes' last paper. This does, I think, illustrate a point which is interesting, and that is that a concentrated interest in thyroids will reduce the mortality, and a limited number of men performing thyroidectomies will reduce the mortality still further. As long as many men do only a few thyroidectomies, we will never reduce the mortality to where we wish it.

In a recent paper read before the American College of Surgeons at Philadelphia, I presented the work of Dr. E. C. Bartels in our Clinic on the hippuric acid determinations of liver function. We have demonstrated, I think beyond any reasonable doubt, that in the severe stages of hyperthyroidism, there is a diminished liver function as demonstrated by hippuric acid tests; that during the period of preoperative preparation, this is elevated; that during the period of postoperative reaction, the liver function, in terms of hippuric acid determinations, is again diminished and that when the basal metabolism returns to normal and the patient is relieved of his hyperthyroidism, hippuric acid determinations reach their normal range of 2.8 to 3 Gm. We do not believe, however, that hippuric acid determinations of liver function are of any great value in determining operability. When a patient is so seriously ill from hyperthyroidism that his liver function, in terms of hippuric acid determination, is definitely diminished, there will be many other evidences that should make one assume that the patient falls into the bad risk group.

The selection of bad risks can only be the result of the accumulation of a mass of data which must be assembled and appreciated and based solely upon experience, before one can segregate those patients who have severe grades of hyperthyroidism in whom there is a danger of a fatality, from those with less severe grades in whom one stage operations may be performed safely.

One of the outstanding facts that has been impressed upon us in the selection of thyroid risks is that there never has been, and probably never will be, any substitute for experience.

There are facts which should be presented on this subject, that particularly relate to statistics, and I wish to be careful that no one assumes that these figures are presented in any comparison whatever with the groups already presented. It pleases me very much that Doctors Drennen, Maes and Parsons are doing such splendid work with thyroid states, and I speak of numbers after they have spoken of numbers, only that I may prove the point which I wish to make.

We have now performed 15,300 thyroid operations on 13,000 patients. Of these, 111 died while in the hospital. These include every death that occurred from any known cause, such as those who developed typhoid, measles, or one patient who stumbled on the top step and broke her neck, and so forth. During that same time, 53 patients died while in the hospital upon whom no operation had been performed. Of those who died following operation, 40 per cent died in crisis, 10 per cent of emboli, and 10 per cent of heart failure. During the same time 33 per cent of those who had had no operation died in crisis, 14 per cent of emboli and 15 per cent died of heart failure. This means that almost all of these deaths were avoidable deaths. The Bureaus of Vital Statistics in Massachusets, New York and Pennsylvania have informed me that: During a period of six years, in these three states, they had recorded 5,321 patients who, during this period, had died thyroid deaths, most of them without operation. If we could operate upon 15,000 people with 111 deaths and if 5,321 died in six years in these three states, we must do something about it. Since a third of them died in crisis, they would not have died had they been operated upon earlier. Since 10 to 14 per cent died of emboli, they would probably not have had emboli if they had been operated upon earlier and had not had auricular fibrillation, and since 10 to 15 per cent died of heart failure, most of these hearts would not have failed if they had not had hyperthyroidism over a long period of time.

I wish particularly to urge three points: First, as soon as any patient with hyperthyroidism shows any increase in tensity of his symptoms, one should drop conservative measures and resort to surgery; second, when there are any evidences of an impending crisis, likewise, one should drop conservative measures and resort to surgery; third, auricular fibrillation is accepted altogether too lightly and, in my opinion, should be an immediate indication for surgery in hyperthyroidism. You do not have emboli as a rule until patients' hearts begin to fibrillate. They do not fibrillate as a rule unless they have heart damage and hyperthyroidism. Therefore, if you have a patient with hyperthyroidism and he develops fibrillation, that, in my opinion, becomes an immediate and urgent indication for thyroidectomy.

I would like to say something about total thyroidectomy, and if I do not seem enthusiastic about it, convictions have been arrived at on personally operated cases, and others, now amounting to 27 in number, that we have carefully followed and are being reported. The number of good results in this series is, in our opinion, not sufficient to make us entirely pleased with them. I do not believe that this operation will endure as a much practiced procedure for the following reasons: First of all, any patients who are operated upon for cardiac decompensation by total thyroidectomy have, of necessity, a very limited cardiac reserve; secondly, one must produce a myxedema, and it is accepted that myxedema is an undesirable state for a decompensated heart. Therefore, it is said that these patients should be given thyroid extract until they are maintained at approximately a minus 25 metabolism. We feel the-

oretically, and based upon our experience in this limited number of cases, that this is too complicated a position to maintain with any degree of accuracy, and in the majority of cases of cardiac decompensation upon whom total thyroidectomy has been performed, they have had a return of their decompensation or have died. In the cases of angina pectoris, there have been a few cases who have been distinctly benefited and if a patient is willing to exchange an active state for the sluggish state associated with myxedema, there are undoubtedly certain cases in which the anginal pain can be modified.

Total thyroidectomy is a difficult operation. It is one in which there will be a very definite mortality; it is one in which there has been in many hands a high percentage of complications. It demands the highest degree of technical skill and an intimate knowledge of the anatomy of the thyroid. It, therefore, should not be undertaken lightly. I believe as years go on, it will occasionally be employed for the well selected patient, the selections being made largely by a competent cardiologist in terms of the amount of cardiac reserve. I believe, on the other hand, that as the years do go on, the total number of cases operated upon by total thyroidectomy will, as it seems to me they are now doing, progressively diminish.

DR. URBAN MAES (closing).—Three years ago, after I had read my very pessimistic first paper on thyroid disease before this organization, I thought I would take a train in the opposite direction instead of going home. To my great surprise, I found the New Orleans surgeons rather enthusiastic about the truthful report we had made on the subject, and it is gratifying to find that a sharp drop in the mortality of thyroid surgery has occurred since that time. On the other hand, we must remember that it is going to take surgeons of the South a long time to become proficient in this branch of surgery. We see too few cases. That is proved by the large number of surgeons who handled this relatively small number of patients.

The estimation of liver function is only one factor in the general estimate of the patient with thyroid disease, but it is a very important consideration, and disregard of it will appreciably increase the mortality. Patients with a low liver function run a rather poor chance after thyroid surgery. I want to stress again and again that this is only one factor to be considered, but I also want to stress that it is a most important one, and it is more important, I believe, where experience in thyroid surgery is limited, than where it is extensive. Perhaps, if we give this factor sufficient weight, we may reduce the mortality of thyroid surgery in New Orleans to something approaching the mortality Doctor Lahey achieves in Boston. That is a consummation devoutly to be wished, but one not likely to be achieved, I fear, in the community from which we come.

DR. WILLARD H. PARSONS (closing).—Because he speaks, and rightly so, with the voice of authority, Doctor Lahey's antipathy to the procedure in question will tend to consign total thyroidectomy for cardiac disease to an early, and, we believe, possibly untimely death. Yet careful analysis of his remarks indicates that certain of his conclusions are not different from our own.

No one could maintain that the operation is ideal, and none could deny that for technical and other reasons it should not be lightly undertaken. Likewise it does, as Doctor Lahey remarks, convert an individual formerly alert to a somewhat vegetative existence. At that, such a conversion may be a fate more kindly than its predecessor. In considering the disadvantages incident to the thyroidectomized patient, we must, in fairness, consider also the dis-

advantages to the individual from the disease because of which treatment is undertaken.

We agree with Doctor Lahey that the principal value of the operation is in the management of patients having angina pectoris rather than congestive heart failure. We have said, and we repeat, that total thyroidectomy, admittedly an objectionable procedure, should be reserved for a small, though definite, group. In that small refractory group, meeting the qualifications emphasized by other authors and by ourselves, until some more effective or desirable form of therapy is suggested, we think total thyroidectomy worthy of trial.

Before abandoning a procedure which, if the literature is to be believed, has been of value in rather a high percentage of cases, we must inquire if some therapy, at least equal if not more efficacious, is offered.

FACTORS OF SIGNIFICANCE IN THE PROGNOSIS OF CANCER OF THE STOMACH

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ANY discussion of cancer of the stomach should include some reference to the problem of early diagnosis. Cancer of the stomach in its early stages is so situated that in probably 90 per cent of cases the lesion could be satisfactorily removed, yet the present-operability in the disease is only between 20 and 25 per cent. Of no other form of cancer is this paradox as true. The chief reason for this is the absence of definite symptoms in the early stages of the disease, and the hope of any advances in detecting the disease in this operable stage rests on the alertness of the layman and the physician to the facts that cancer of the stomach is by far the most common organic disease of the stomach and that there are methods of determining whether or not the disease is present. The skilled roentgenologist, in 95 per cent of cases, can visualize organic lesions of the stomach, although concerning approximately 20 per cent of these a definite opinion as to the nature of the lesion is not warranted. This inability to distinguish between some of the lesions is also the experience of the surgeon, and it is frequently true that not until microscopic examination is possible is the true nature of the lesion revealed. To what extent the gastroscope, in skilled hands, can improve this situation remains to be seen, but it seems reasonable to believe that any method which satisfactorily will allow visualization of the entire mucosal area of the stomach, holds great possibilities for development.

The curability of cancer of the stomach by surgical removal of the growth has been well established. When the growth and the regional lymph nodes can be thoroughly extirpated, five year cures* occur in about 30 per cent of cases; this is based on 18 per cent of five year cures when lymph nodes are involved and 48 per cent of five year cures when lymph nodes are not involved. Such facts, in contrast to the absolute hopelessness of the disease with any other method of treatment, constantly should emphasize the importance of developing every means for recognition of the disease while the growth still can be removed.

Accuracy in prognosis in disease is properly interpreted by the layman as an evidence of professional experience and knowledge. In cancer of the stomach, this is particularly true and whether or not the diseased tissue has been removed, relatives and friends can much more easily adjust themselves

* I have selected the five year period after operation as the time to estimate apparent cure for the reason that consideration of the charts will disclose the important fact that after five years, the death rate of those who have undergone gastric resection for carcinoma is approximately the same as that of the general population group of similar age and sex.

to the probable course of events, than to great uncertainty. When the condition is inoperable, any information which can be given as to the expectation of life, the nature of the symptoms which probably will mark the course of the disease, and what can be expected from the treatment of these symptoms, is most gratefully received. Similarly, in those cases in which the growth can be removed, or some palliative procedure can be carried out, the family should be informed of the facts on which prognosis is based and it is with a consideration of these facts that the remainder of this paper is concerned.

The findings to be reported here, as they are related to prognosis, are based on a series of 4,793 cases of gastric carcinoma in which operation was performed in The Mayo Clinic in the period 1906–1931. In 2,112 of these cases the growth could be removed either for palliation or in the hope of cure. This is a rate of operability of 45 per cent, based on the patients subjected to exploration, and of only 19 per cent, based on the number of patients concerning whom a diagnosis of cancer of the stomach was made. In 17 per cent, palliative gastro-enterostomy was done, and in 38 per cent, neither resection nor gastro-enterostomy seemed advisable (Table I). The expectation of life in the group in which exploration revealed the disease too advanced for either gastric resection or gastro-enterostomy was five months. In the group in which gastro-enterostomy was performed, the expectation of life was only one month more, or six months, and the mortality relative to the operation was 11 per cent.

TABLE I

CARCINOMA OF STOMACH
HOSPITAL MORTALITY FOR CERTAIN MAJOR OPERATIONS
1906 TO 1931, INCLUSIVE

Operation	Number	Hospital Mortality	
		Number	Per Cent
Partial gastrectomy	2,112	295	13.9
Gastro-enterostomy	833	96	11.5
Exploration	1,848	64	3.5
Totals	4,793	455	9.4

In the cases in which the growth was removed, the hospital mortality was 13.9 per cent (Table I). As is evident in Chart 1 the mortality seems to vary with the attitude of the surgeon toward the cases in which the disease is greatly advanced, for in some years the mortality has been as high as 23 per cent and in others as low as 8 per cent (120 cases with ten deaths). A low mortality is chiefly dependent on proper preparation of the patient for operation and meticulous attention to all those details which lessen the likelihood of development of the two chief causes of death in such cases; namely, peritonitis and pneumonia.

Many factors may be taken into consideration in estimating prognosis when the growth can be extirpated: namely, the age of the patient; the duration of the symptoms; the gastric acidity; the size, situation and extension of the lesion (into the serosa, lymph nodes, and so forth), and the pathologic characteristics. Although some of these factors proved to be of little significance, they are at least interesting and contribute to better knowledge of the basis of prognosis.

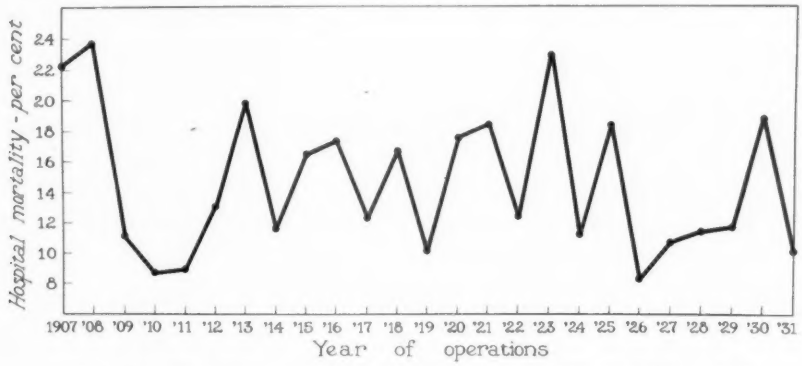

CHART 1.—Hospital mortality in operations for gastric carcinoma (1907–1931).

In so far as age is concerned, it is shown in this series that the percentage of five year survival in the disease is higher among the older patients (33 per cent in the age group of 45–54 as contrasted with 25 per cent in the 35–44 group) (Chart 2).

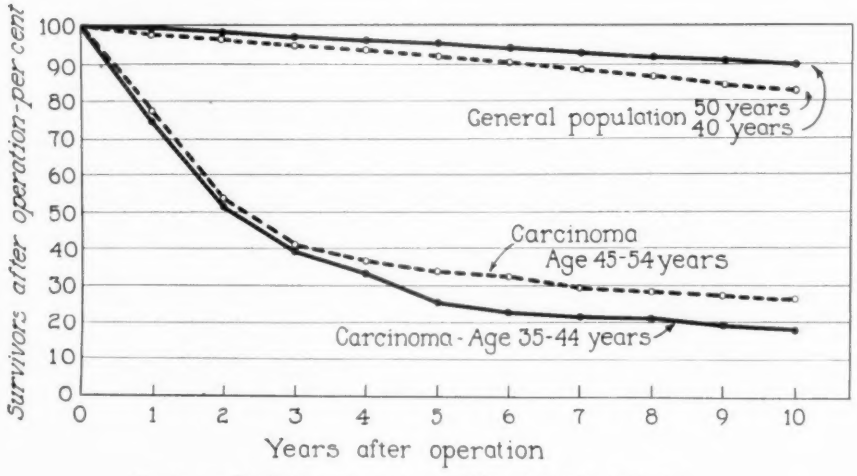

CHART 2.—Significance of age in curability of gastric carcinoma.

The length of history disclosed the interesting fact that five year survivals were more frequent among those cases in which gastric symptoms were of longer duration, for among those patients whose symptoms had been present for 12 months or more, 35 per cent lived five years while among those

whose symptoms had been present for six months or less, 25 per cent were alive and apparently well at the end of five years (Chart 3).

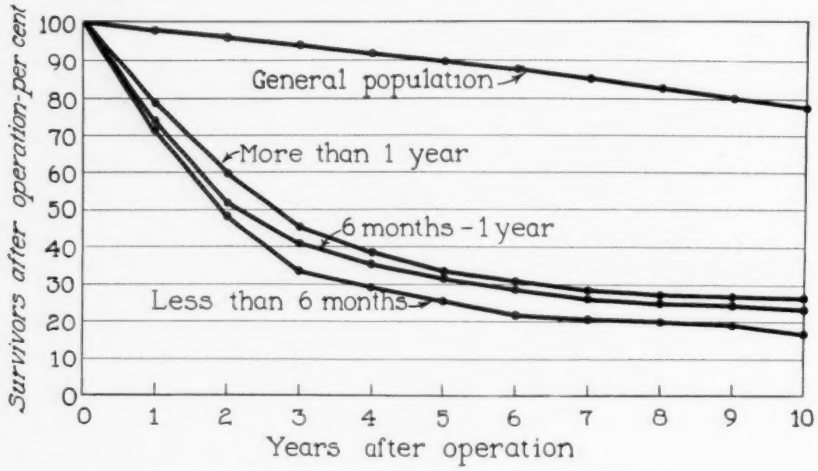

CHART 3.—Significance of duration of symptoms in prognosis following gastric resection for carcinoma.

In respect to the significance of secretory function of the stomach, I always had thought that the patient who had normal secretory activity would not have as good prospect of cure as one with less active function; yet this series shows the reverse, for among those patients whose secretory function was approximately normal, the five year survivals were 15 per cent more than among those whose secretory function was markedly diminished or absent (Chart 4).

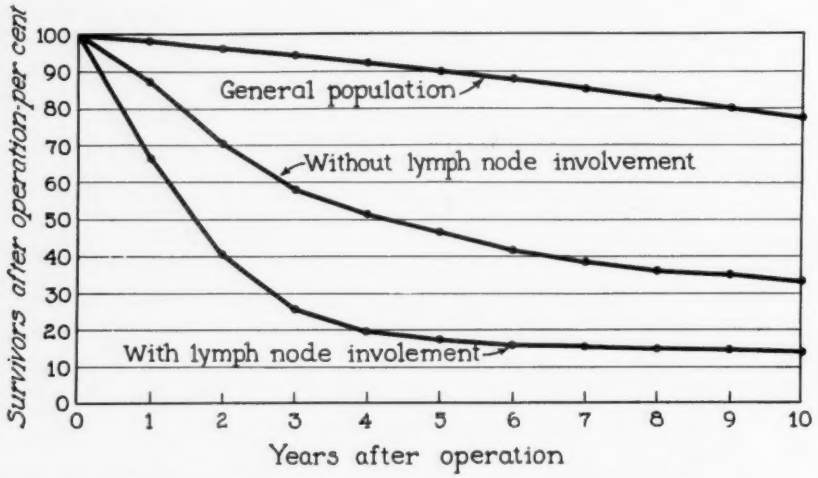

CHART 4.—Significance of gastric acidity on postoperative results.

Investigation of survival, based on the size of the lesion, disclosed the curious fact that there was greater expectation of life among patients who had the larger lesions than among those who had the smaller lesions. This

is probably attributable to the fact that the smaller lesions are more likely to be of a penetrating character and also of a higher degree of malignancy than the larger lesions. Survival of patients who had the larger (60 sq. cm.) lesions was compared with survival of patients who had the smaller

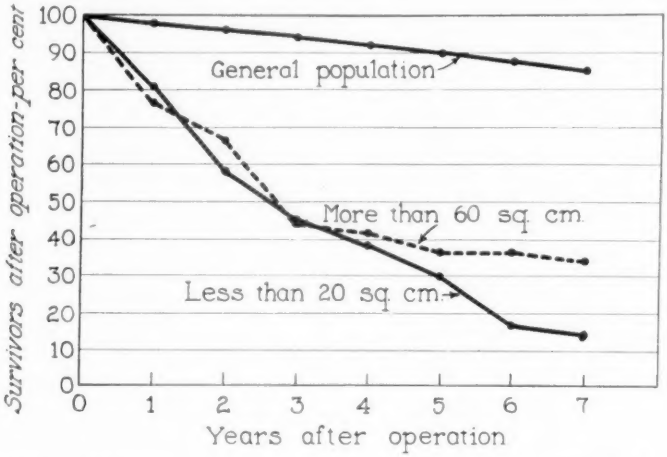

CHART 5.—Influence of size of lesions on results of gastric resection for carcinoma.

(20 sq. cm.) lesions; five years after operation, about 10 per cent more of the former than of the latter were living (Chart 5).

The situation of the lesion is of significance and in this series (Chart 6), the observation of others is confirmed; namely, that the nearer the lesion

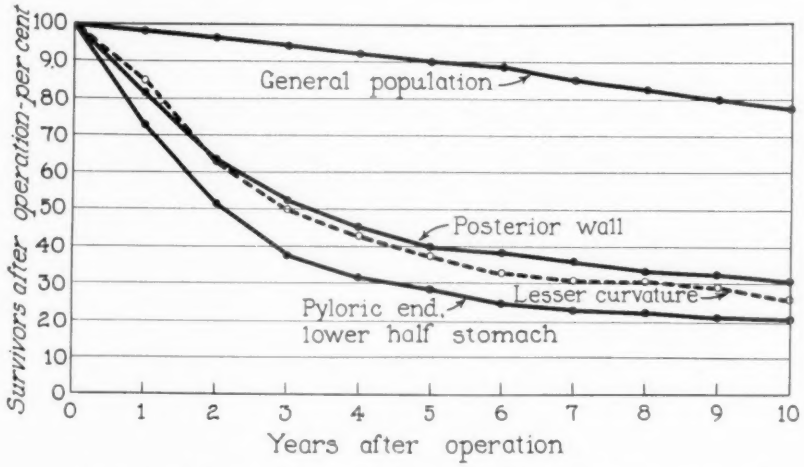

CHART 6.—Significance of site of lesions on results following gastric resection.

is to the pylorus, the more difficult it is to cure, and removable lesions in the body of the stomach are accompanied by a distinctly higher rate of survival than are those near, or involving, the pylorus (40 per cent compared to 28 per cent). This may be attributable to the fact that regional lymphatic

structures are more easily removed with thoroughness when they are in the former situation than in the latter, and also to the fact that not enough attention has been given to the importance of removing, in the resection, a segment of the first portion of the duodenum. It has been shown that although gross involvement of the duodenum is exceedingly rare in cases of cancer of the stomach, microscopic invasion not infrequently can be demonstrated.

The extension of the lesion has great significance in prognosis. Involvement of serosa was shown by Gray to decrease the possibility of cure by approximately the same per cent as involvement of lymph nodes, which, as with cancer in all situations, lessens curability to a marked degree. Five years after operation for cancer of the stomach, as has been said earlier in this paper, 18 per cent of patients whose lymph nodes are involved and 48 per

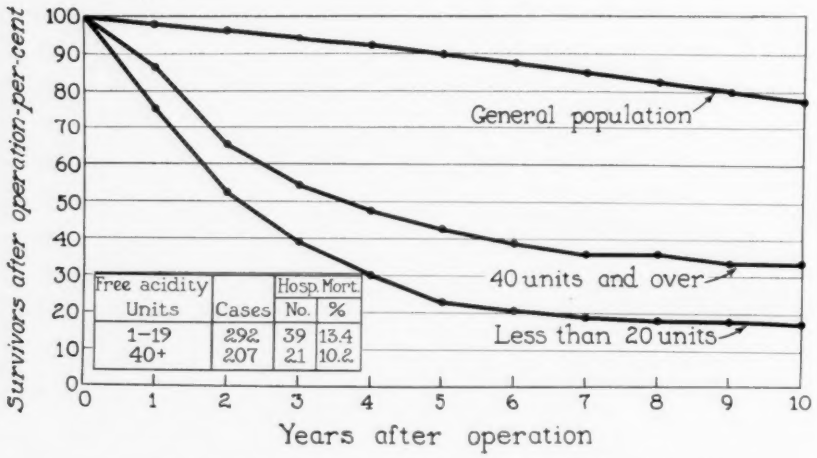

CHART 7.—Significance of involvement of lymph nodes on curability of carcinoma.

cent of those whose lymph nodes are not involved are alive. The difference then is 30 per cent (Chart 7).

The most accurate prognostic information obtainable in this series proved to be grading of malignancy by the method of Broders in which the degree of cellular differentiation is recorded as of Grades 1, 2, 3 and 4. Of patients whose carcinomata are of malignancy graded 1 and 2, 63 per cent are alive five years after operation, and ten years after operation 55 per cent of patients of this group are alive. These data can be contrasted with the fact that of patients whose carcinomata are of malignancy graded 3 and 4, only 20 per cent are alive five years after operation. These results again substantiate the fact that grading of malignancy stands first in importance in prognosis (Chart 8).

The coordination of these various factors has in our experience at the Clinic added definitely to accuracy in prognosis. This investigation also has supported the contention of surgeons that the surgical treatment of cancer

of the stomach can and does accomplish more than is recognized, and that constant repetition of this fact is the best approach to earlier recognition of the disease.

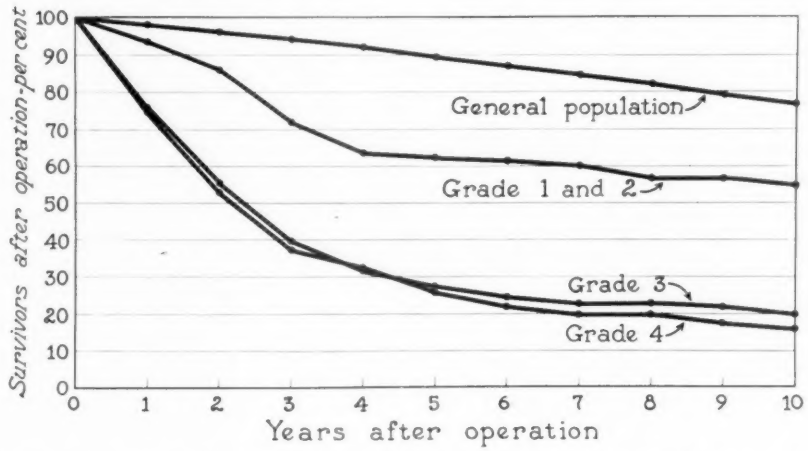

CHART 8.—Significance of histologic grading (Broders) on prognosis following gastric resection for carcinoma.

DISCUSSION.—DR. J. M. T. FINNEY (Baltimore, Md.).—I wish to express my own personal satisfaction and am sure I speak for the entire group, when I thank Doctor Balfour for bringing out so graphically and clearly certain points that from his wealth of material he has been able to bring to the fore, and for emphasizing a number of other points of great practical importance.

DR. HAROLD L. FOSS (Danville, Pa.).—In discussing Doctor Balfour's paper may I approach the subject from a slightly different angle? In his experience at Rochester, 19 per cent of the patients were not in condition to be aided by any form of treatment. It is as important as ever to keep before the public the paramount necessity of early diagnosis in malignant disease. It is estimated, in Pennsylvania, that there are over 100,000 patients suffering from some form of neoplastic disease. The United States census bureau reports that 43 per cent of all Americans live in rural districts. Those who work in medical centers, serving rural sections, have found it to be true that rural patients invariably come much later for treatment than is the case with the patient of the metropolitan areas. In discussing a paper recently presented by Doctor Lahey covering some 200 cases of gastric carcinoma, I reviewed an equal number of my own. Doctor Lahey's patients came from a large metropolitan area, mine from the rural districts of central Pennsylvania. While 30 per cent of the city patients showed weight loss, 70 per cent from the rural sections had it as a chief symptom. Thirty per cent from the Boston area were vomiting; 70 per cent from the rural district. About 5 per cent of Doctor Lahey's patients had had hemorrhages; about 30 per cent of ours. In the patients coming from the city, 25 per cent were operable; in my series, but 5 per cent. In the inoperable group there was no operation whatever in 36 per cent of the urban cases; in the rural group, none in 57 per cent. In only 11 of the 200 rural patients was it possible to perform a resection. Fifteen per cent of all patients admitted

to our hospital have some form of neoplastic disease, nearly always in an advanced form. Our patients with carcinoma of the breast wait 15 months before seeking aid; those with carcinoma of the colon 11 months from the onset of the symptoms. Those with gastric carcinoma delay, on the average, eight months.

In our state, and I think this is typical of all states, there is a large group of sufferers destined to be treated far too late. In the rural districts these patients have not been so much in need of surgery as of education. Nearly 50,000,000 of our people live in the country and they are the ones not being reached by the groups trying to do the things Doctor Balfour says are so important. As was stated in the paper, surgery of the stomach has probably reached as high a degree of technical perfection as is possible and it is not likely that much greater progress will be made by the roentgenologists or diagnosticians in their fields. More can be accomplished only if we can aid these agencies in the education of those people who are likely to develop cancer, or who have it, and who know nothing about the disease. I recently had as a patient the director of the Public Works Administration for Pennsylvania. He informs me that in our state the W. P. A. is spending over $20,000,000 a month—on roads and airports and bridges, *etc.* I asked what was being done regarding lay education on medical matters and he told me "nothing."

The mortality of cancer will not be decreased, and surely the operability of cancer of the stomach will not be increased, until patients can be seen far earlier. While the Society for the Control of Cancer, the American College of Surgeons, cancer commissions and other groups are, to a certain extent, reaching the patient of the larger medical centers, patients constituting nearly 50 per cent of our American population, those living in the rural sections, know nothing of this program of education or of the need for it.

Four billions of Federal money is being spent on all sorts of projects, most of them, no doubt, meritorious. Why not take 25 millions of this, say but one month of Pennsylvania's allotment, for lay education dealing with the menace of malignant disease, its prevention and early eradication, especially lay education among our rural patients? No one could doubt but that thousands of lives would be saved and incalculable suffering prevented.

CHRONIC OBSTRUCTION AND DILATATION OF THE DUODENUM

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SINCE the report of Bloodgood,[2] in 1907, of a case of chronic dilatation of the duodenum, considerable interest and much controversy have been aroused over the significance of the dilated duodenum and the question as to whether in the condition designated "chronic duodenal ileus" we have a true pathologic and clinical entity. Balfour,[1] Bloodgood,[3] Duval, Roux and Beclere,[6] Halpert,[7] Kellogg and Kellogg,[10] Stavely,[14] Wilkie[16] and others have reported clinical and pathologic observations supporting the conception that chronic duodenal obstruction is a definite pathologic and clinical entity and amenable to surgical treatment.

In recent years there has been considerable controversy as to the value of surgery and it has been said, "As time passes, the superior mesenteric artery pressure theory will fade until it disappears altogether from our horizon."[13]

That other forms of dilatation of the duodenum occur is more than probable and is no argument against the existence of the one with which we deal here.

Imperfect rotation of the colon in fetal life, which brings the superior mesenteric vessels to lie across the duodenum, and imperfect fixation of the colon are found most frequently in asthenic and visceroptotic types, and it is in this class of individuals that duodenal ileus is most frequently encountered.

The tolerance of the small intestine to partial interference with the onward progress of its fluid contents is well known and it is easily conceivable that a partial duodenal obstruction may exist for years without symptoms sufficiently pronounced to recognize the lesion clinically until, finally, a complete obstruction supervenes with symptoms too obvious to be overlooked.

The reported results vary with different surgeons. Some report excellent results[9] but surveys by others show complete cures in only about 50 per cent of the cases.[8, 11, 12, 17]

It is our opinion that the reported poor results are the results of the application of sound surgical principles in the absence of a definite obstructing mechanism.

The evidence submitted in the following case report sustains the conception of chronic duodenal obstruction as a clinical entity amenable to surgical treatment.

Case Report.—G. W., white, female, age 18, complained of nausea and vomiting for six weeks. In the beginning the complaint was characterized by nausea only. After a few days vomiting ensued. The vomitus was described as bilious—large quantities of bile-stained fluid. Confinement to bed and liquid diet gave a measure of relief for ten days and then she began vomiting everything taken into her stomach. At times food taken 24 hours previously was noted. At no time during these six weeks had she suffered pain.

The past history is of great interest and perhaps significant. At three weeks of age, because of persistant vomiting, she was taken to the late Doctor Tuholske of St. Louis, whose diagnosis was "pylorospasm" and whose treatment consisted of stomach washings and forced feeding by means of a tube for a period of six weeks. At the end of this time, her condition being improved, she was put back on the breast and did fairly well, with the exception of occasional spells of vomiting, until her second summer, when she suffered from "colitis" for eight weeks. This attack of colitis was characterized by much vomiting and alternating constipation and diarrhea. At six years of age she was sick for a year and lost much weight because of frequent attacks of nausea and vomiting. The vomitus consisted of bile-stained fluid and food particles. During this sixth year of life numerous and varied examinations by many physicians resulted in no definite diagnosis. For the past 11 years she has had no definite attacks of vomiting, but, as her mother describes her condition, "She has been a poor eater, many foods disagreeing with her, producing a sense of fulness and much belching." The gaseous eructations have been a source of great embarrassment to her. All her life she has been undernourished and frail and a source of anxiety to her parents.

This is a fairly accurate history of the patient until the onset of the present complaint some six weeks previous to her admission to the hospital.

Physical Examination.—Patient is an asthenic, visceroptotic young woman, five feet, two inches in height, weighing 98 pounds, with a normal temperature, pulse 70, blood pressure 95/70. The heart and lungs are normal. The abdomen is flat, muscles flaccid, and there is tenderness over Mayo-Robson's point. There is marked ptosis of the right kidney. Pelvic examination reveals a retroverted uterus. Examinations of blood and urine were reported normal. Basal metabolic rate + 13.

Roentgenologic Examination.—(Dr. William D. Anderson).—Fluoroscopy: The lungs appear normal. The heart is small and in the midline. The breathing is costal in type and expansion is about equal on the two sides. There is moderate scoliosis of the dorsal spine with convexity to the left and compensatory scoliosis of the lumbar spine with convexity to the right. There is tilting of the pelvis to the right. The esophagus is normal. The cardiac orifice is normal. The stomach is the long fish-hook type and when filled, extends well down below the brim of the pelvis, the greater curvature reaching a line joining the lower ends of the two sacro-iliac joints. There is a moderate amount of fluid and mucus present in the fasting stomach and the barium meal can be seen falling through this supernatant layer of fluid. The tone of the stomach is extremely poor. Peristalsis is almost entirely absent, only an occasional wave of slight depth being noted. Mobility of the stomach is normal. Motility is slow. The pylorus is patent and shows no spasm. There is no evidence of defect or niche in the gastric outline. The gastric rugae are easily brought out but are not increased in size. The duodenal cap fills out well, is triangular in outline and shows no evidence of defect or spasm. There is no irritability. The second portion of the duodenum is dilated, with increase in the rugal markings and an extreme writhing and to-and-fro motion of the barium meal. About three inches from the cap the meal meets an obstruction, producing the dilatation and past which the meal cannot actually be seen to pass, although small amounts of barium are passing, as it can be seen farther down in the jejunum. In the prone position with the patient turned slightly to the right, this dilated portion of the duodenum appears flattened as if by pressure of an enlarged or

ptosed liver. When the patient is turned slightly to the left, this flattened appearance disappears. The definite cause of the obstruction cannot be determined under the fluoroscope.

Roentgenography.—Immediate.—Vertical Position: shows the body of the stomach well down in the pelvis, as seen on fluoroscopy. The stomach is dilated and atonic. The barium meal puddles in the dependent portion. Traces of barium may be seen adhering to the mucosa of the prepyloric region, the duodenal cap and the proximal part of the second portion of the duodenum. There is some barium collected in the distal end of the second portion of the duodenum at the apparent point of obstruction. Horizontal position, patient on back, shows the body and cardiac end of the stomach filled and dilated but otherwise normal. Traces of barium in the prepyloric region and cap. Prone Position: Stomach is well filled and still atonic. No evidence of defect or niche in the gastric outline. Duodenal cap well filled and normally outlined. The second portion of the duodenum is filled and dilated for a distance of about four inches beyond the cap, and is flattened on the right side due to pressure on the liver, which

FIG. 1.—Roentgenogram before operation, showing the dilated and obstructed first and second portions of the duodenum to the right of the midline.

FIG. 2.—Appearance of the stomach and duodenum at the end of six hours, showing the gastric and duodenal retention.

is very low, extending four fingers' breadth below the costal margin. Traces of barium may be seen in the third portion of the duodenum (Fig. 1).

Two and one-half hours.—The stomach is still half full and shows no better tone than on previous film.

Five hours.—The stomach is approximately one-third full. Duodenum is still dilated and filled with barium to the distal part of the second portion.

Six hours.—The stomach is about one-fourth full. The duodenum does not show as much dilatation at this time as before. However, there is still barium present in the second portion, which ends abruptly at a point about four inches from the cap. Traces of barium are seen in the third portion, which does not appear to be dilated. Barium is scattered throughout the remainder of the small intestine, which appears normal, except for the extreme ptosis (Fig. 2).

Twenty-four hours.—Patient vomited since last observation. No barium in stomach or upper intestine. There is irregularly scattered barium throughout the colon, which is not filled well enough for accurate study. There is some barium in the terminal ileum. The appendix cannot be definitely visualized.

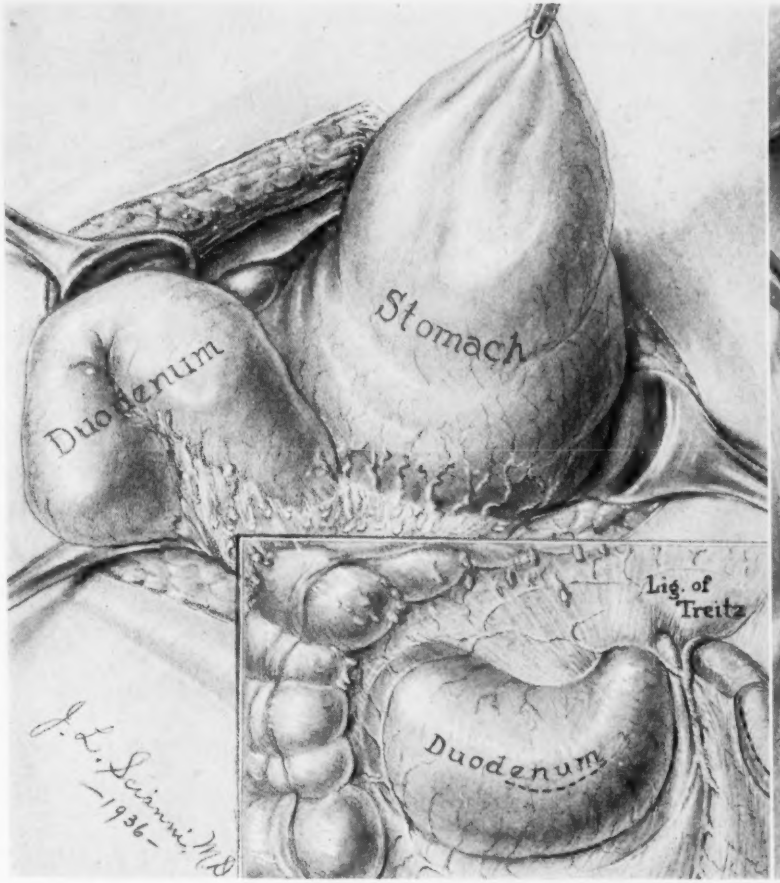

FIG. 3.—The dilated stomach and mobile duodenum with marked dilatation of the first and second portions. (Insert) Dilated third portion of the duodenum shining through transverse mesocolon. Dilatation limited at point of crossing of superior mesenteric vessels and drag of mesocolon. The small empty jejunum.

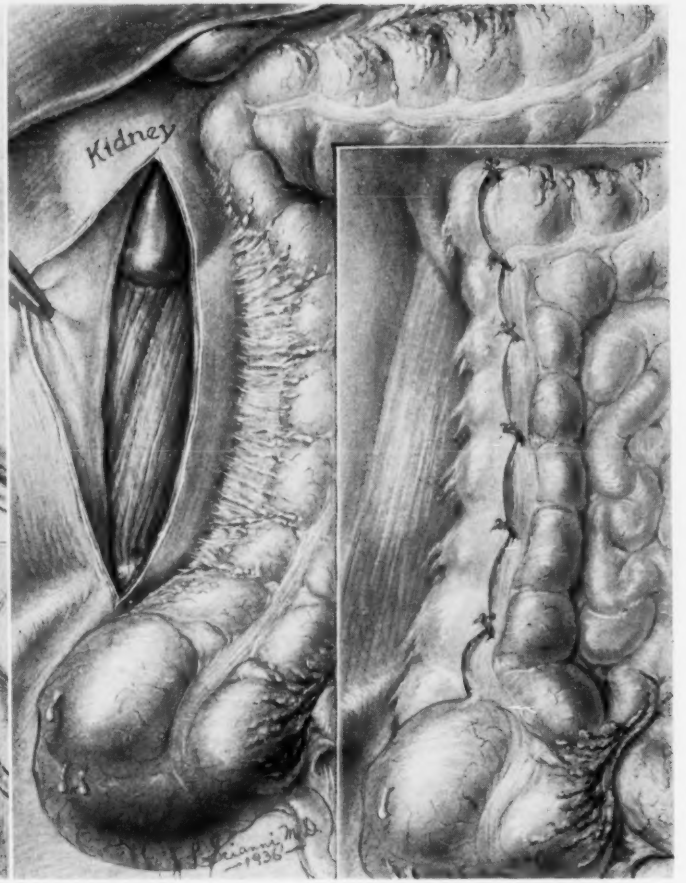

FIG. 5.—Mobile colon with its mesentery and Waugh colopexy.

Roentgenologic Diagnosis.—(1) Chronic partial duodenal obstruction with dilatation, the point of obstruction being apparently four or five inches distal to the pylorus. (2) Dilatation and atony of the stomach. (3) Generalized visceroptosis, extreme degree. (4) Scoliosis of the spine.

Operation.—On December 2, 1935, with the preoperative diagnosis of chronic duodenal obstruction with dilatation, the patient was subjected to operation. Under ether anesthesia, through a right rectus incision, the following gross findings were revealed (Fig. 3): The stomach dilated; walls thin, pylorus patulous, admitting two fingers. The duodenum mobile, attached by a mesentery, the first and second portions dilated to three times the normal size. On raising the transverse mesocolon, the greatly dilated third portion of the duodenum was seen bulging through the transverse mesocolon. This dilatation of the duodenum extended to the point of crossing of the superior mesenteric artery but at this same point there was also considerable drag of

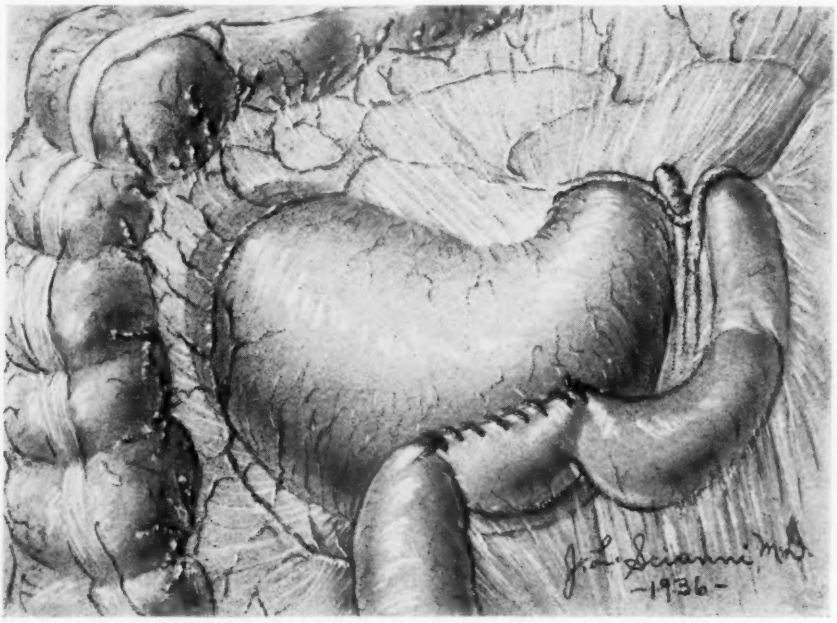

FIG. 4.—Submesocoloic duodenojejunostomy.

the mesocolon, which was relieved by lifting up a mobile colon. The jejunum was empty and smaller than normal by one-half the normal circumference. On pulling up the mesentery, the distended third portion was seen to empty into the jejunum, filling it. The ascending colon was mobile, attached by a mesentery. It could be lifted out of the abdominal cavity. The appendix was normal. The right kidney was ptosed. The uterus was retroverted. The liver was low, extending four fingers' breadth below the costal arch. The gallbladder was normal.

A submesocolic duodenojejunostomy (Fig. 4) was performed, by the clamp method, between the dilated third portion and the jejunum, the mesocolic opening being between the superior mesenteric artery and the right colic artery; also an appendicectomy, and a colopexy, after the technic of Waugh[15] (Fig. 5).

The patient made an uninterrupted recovery and on the fifth postoperative day was taking a light diet and enjoying it. She was discharged on the twelfth postoperative day, at which time she was taking a liberal diet, with no digestive complaint.

One year has passed since this operation was performed. The patient has been

FIG. 6.—The stomach and duodenum one month after operation. The arrows indicate the passage of barium through the operative opening between the third portion of the duodenum and jejunum.

FIG. 7.—Six hour roentgenogram one month after operation. Note that stomach is completely empty and barium meal is now in the colon and terminal ileum.

FIG. 8.—Twenty-four hour roentgenogram one month after operation. The colon is well filled and only slightly prolapsed.

well. She reports that she has had no indigestion; her weight is 106 pounds; and she has completed another year of college work, participating in all college activities.

COMMENT.—The very definite roentgenologic findings of duodenal obstruction and dilatation, on which the preoperative diagnosis was made, may be seen readily in Figs. 1 and 2. These two findings, together with the writhing motion proximal to the obstruction, noted on fluoroscopic examination, and the delayed emptying of the stomach and duodenum, complete the characteristic roentgenographic picture of chronic duodenal obstruction.[4]

The point of obstruction was demonstrated roentgenologically to be in the lower, second portion of the duodenum. This corresponded to the operative finding of mesocolic duodenal compression by the middle colic artery and explains why the obstruction, by compression from the superior mesenteric artery, was not visualized.

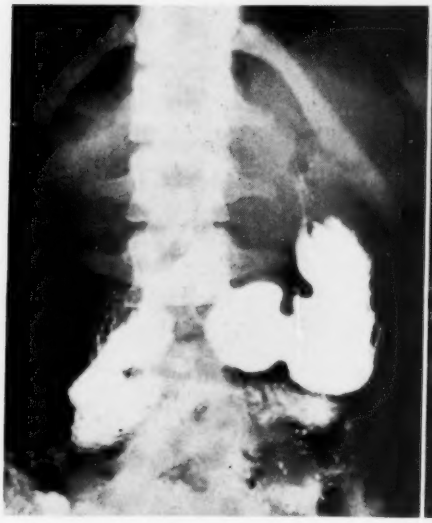

FIG. 9.—Roentgenogram of stomach and duodenum one year after operation, showing good emptying.

FIG. 10.—Six hour roentgenogram one year after operation. The stomach and duodenum are practically empty at this time.

A second series of roentgenologic studies was made one month after operation. This clearly demonstrated the restoration of normal function. The duodenojejunostomy is well depicted in Fig. 6. The stomach and duodenum are completely empty at the end of the six hour period (Fig. 7). At the end of 24 hours the colon is well filled and only slightly prolapsed (Fig. 8).

A check up roentgenologic examination at the end of one year postoperatively reveals the stomach and duodenum still emptying freely (Fig. 9). The slight gastric residue noted on the six hour film (Fig. 10) is probably explained by the composition of the barium meal—milk and barium instead of water and barium, as used in the two former examinations.

The findings in this case demonstrate that the obstruction of the duo-

denum was due to a combination of two factors; namely, arteriomesenteric and arteriomesocolic strain. "The conception of the anatomical conditions underlying the arteriomesenteric and the arteriomesocolic occlusion of the duodenum may or may not be a pure academic discussion."[5] The findings in this case were very definite. The clinical history and the operative findings strongly support the theory that the obstructing mechanism was present from birth—the result of a developmental defect, an imperfect rotation and fixation of the colon—and that its presence was more or less manifest throughout the 18 years of this patient's life. The history, the clinical findings, the roentgenologic observations and the gross findings at operation definitely stamp it as a clinical entity, readily diagnosed.

The dogmatic negation of the "superior mesenteric artery pressure" theory and the substitution therefore of the indefinite terms "neurosis"[6] or "neuromuscular derangement"[13] are not conducive to sound thinking and are not in accord with authenticated clinical observations or clinical end-results.

The operative procedure of duodenojejunostomy supplemented by a Waugh colopexy fulfilled the mechanical indications for relief in this case. That this relief has been realized is evidenced by the restoration of this patient to a normal life.

CONCLUSIONS

(1) Chronic duodenal obstruction with dilatation of the arteriomesenteric type does exist as a clinicopathologic entity.

(2) It is of congenital origin, resulting from the developmental defect of improper rotation of and fixation of the colon.

(3) It can be positively diagnosed by a careful consideration of the history, the physical examination and roentgenologic examination.

(4) The operation of duodenojejunostomy combined with colopexy, after the technic of Waugh, fulfills the surgical indications for relief.

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THE TECHNIC OF CLOSING PERFORATED ULCER OF THE DUODENUM

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IT IS evident that writers on the treatment of perforated duodenal ulcer have found its closure difficult and liable to leak or obstruct the duodenum. They speak of sutures cutting through the friable edges of the ulcer, and of the impossibility of closing large ulcers, which must be drained or provided with a precarious covering of gallbladder or omentum. Deaver[1] is the only writer I have found who states without qualification that all perforations can be closed by suture. Rather significantly, he advocates gastro-enterostomy in all cases. Guthrie and Sharer,[2] in a recent article, report the use of a row of through-and-through sutures of catgut, reinforced by a second row of catgut sutures or a purse-string suture of silk. They must have observed that this method of closure often narrowed the lumen of the duodenum, for they remark that "it is surprising how much the duodenal lumen may be encroached on without producing obstructive symptoms." Kerr[3] advocates closure with a continuous suture placed at right angles to the long axis of the bowel to minimize obstruction. In practically every discussion of this subject in text-books or elsewhere, the probability of duodenal obstruction seems to be foremost in the mind of the author. This explains the almost endless controversy over whether gastro-enterostomy or pyloroplasty should be performed after closure of the perforation. Perhaps the best summary of current methods of treatment, as well as the best conception of the possible difficulties and complications of the closure, is contained in the following advice by Moynihan,[4] published in 1928: "In duodenal ulcers, where the rent is small and surrounding induration absent or of very limited extent, closure of the perforation is all that is required.

"In duodenal ulcer where the rent is larger and induration more extensive, one of the two courses may be followed: (a) The ulcer may be infolded and gastro-enterostomy or gastroduodenostomy performed or: (b) The rent may be enlarged, the ulcer excised, and a plastic operation which secures an enlargement of the passage from the stomach may be performed.

"In duodenal ulcer where the gap is very large and induration extensive, one of the two courses may be followed: (a) The ulcer may be closed as much as possible, and the gap filled by a plug of omentum, gastro-enterostomy being performed or: (b) The opening may be used for the performance of temporary duodenostomy. Both methods are unsatisfactory, and are only to be used in an emergency."

It would seem that a method which does away with most of these difficulties and complications is worthy of trial. The authors have used the technic herein described for about 10 years. It provides a leak-proof and

nonoccluding closure of even the largest perforations. It can be easily and quickly performed.

Operative Technic.—A good incision, which has been previously described by others and which permits of a good exposure of the duodenum and gives a firm wound, begins at the right costal border about two inches from the midline, and extends vertically downward for about five inches. The structures of the abdominal wall, down to the aponeurosis of the transversalis muscle, are divided in the line of incision, and then retracted so as to give a wide exposure of this aponeurosis, which is now penetrated by separation instead of division of its fibers. Since this is the strongest layer of the abdominal wall, failure to close it securely predisposes to evisceration and hernia. It cannot be closed satisfactorily if its fibers are cut, because sutures pull out between the ends of the severed fibers. This incision is easily closed and makes dehiscence of the wound almost impossible, even if it becomes infected.

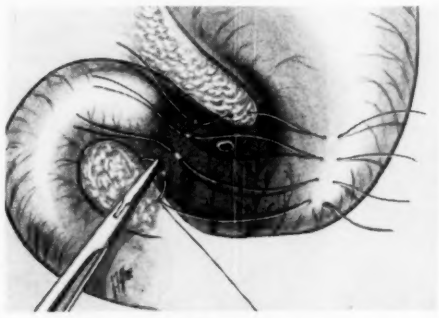

FIG. 1.—Showing the insertion of the sutures into the stomach and duodenum.

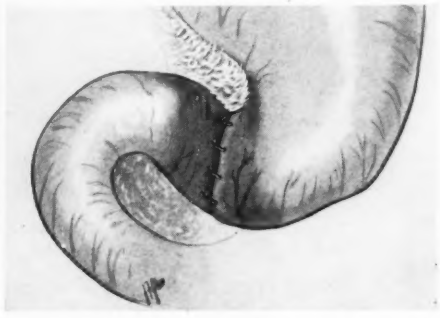

FIG. 2.—Showing the appearance after the sutures have been tied.

The edges of the wound are retracted, and the gastric contents removed from the peritoneal cavity by thorough and systematic aspiration, done as far as possible under direct guidance of the eye. Sponging is not used. The duodenum is now exposed and the perforation located. In our experience, it has been situated almost always near the pyloric sphincter and on the anterosuperior surface of the duodenum. It is always surrounded by a zone of friable tissue, which affords a very unsafe hold for sutures.

Several silk sutures, which pick up the stomach wall about one to two inches from the pylorus, and the duodenal wall, distal to the ulcer, where they can obtain a firm hold, are now inserted, but not tied until all have been placed. These are so placed that when tied they will pull the anterior surface of the stomach over the perforation and close it completely without tension on the sutures (Figs. 1 and 2). The suture line is strengthened where necessary with additional sutures, and then covered completely with omentum, which is sewed in place.

When we first began to use this method, we wondered if it were applicable to the closure of perforations in large calloused ulcers involving the

rear and left lateral surface of the bulb. Experience, however, has shown that these perforations can be easily closed by it, better, I think, than in any other way. If the posterior surface of the bulb above the ulcer is freed, then the sutures can be passed through healthy duodenal tissue, and when tied will pull the stomach wall securely around and over the perforation. The closure can be made more secure by passing the uppermost suture through the gastrohepatic omentum as this suture passes from stomach to duodenum.

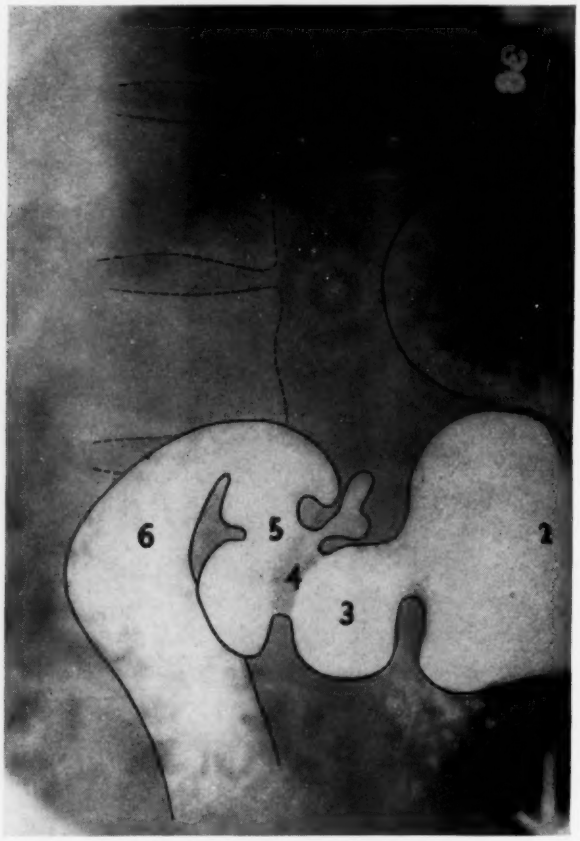

FIG. 3.—Outlined roentgenogram showing the nature of the deformity resulting from the procedure suggested. Note absence of obstructions and of any pinching of the prepyloric part of the stomach. (1) Cardia. (2) Fundus. (3) Pylorus. (4) Pyloric sphincter. (5) Deformed duodenal bulb. (6) Second portion of duodenal bulb.

The superior edges of the duodenal bulb and prepyloric part of the stomach form in most cases a somewhat acute angle with its apex at the upper margin of the pylorus. The anterosuperior surfaces of these parts are not far distant from one another. They can therefore usually be sutured together without much tension.

For a while we feared that the operation would produce a pouch of stomach wall above the pyloric sphincter in which food might stagnate. Roent-

genologic studies, however, have shown this fear to be groundless, and that any pouching of the stomach wall which may be present is too slight to cause dysfunction.

It is a great merit of the operation that it makes unnecessary, in nearly all cases, the performance of gastro-enterostomy or pyloroplasty in addition to closure of the perforation. If the ulcer is not calloused, and is already causing some obstruction, this will seldom be so nearly complete as to prevent the recovery of the patient from his peritonitis, and his survival until the operative relief of his obstruction can be undertaken with comparative safety.

This statement is especially true since the introduction, as a routine postoperative treatment, of continuous gastric lavage and the intravenous administration of sufficient salt solution and glucose.

If, despite expectations to the contrary, the obstruction does prove complete, then the patient, if he recovers from his peritonitis, can be fed until his nutrition is excellent through a Witzel jejunostomy performed under local anesthesia. I have often treated patients in poor condition because of pyloric obstruction in this way, and have fed two patients having a perforated duodenal ulcer similarly. It is to be noted that a patient with complete obstruction because of a calloused ulcer would already, at the time of operation, be in a bad state of nutrition, and not a good subject for more than the minimal amount of surgery.

Though we have spoken with some enthusiasm of this method of closing perforated duodenal ulcers, we do not claim that all of them can be closed by it. We have found it efficient for all perforations except those complicated by an abscess over the duodenum. This is found when the perforation, of long standing, has leaked so slowly that the escaping duodenal contents have been confined by peritoneal adhesions to the neighborhood of the perforation, or to the surrounding region, including the subphrenic space, without contaminating the entire abdominal cavity. Under these circumstances the duodenum will be seminecrotic, and covered all over by a thick, decomposing, fibrinous exudate, which hides it and the perforation from sight. There is probably no method whereby a perforation can be closed effectively in the presence of this complication. I have encountered it on three occasions. Two of the patients, in desperate condition at the time of operation, died. For all three nothing more than drainage was attempted.*

* The patient who recovered was given salt solution and glucose by vein for the first nine days. After that he was fed through a Witzel jejunostomy till his duodenal fistula closed. This was treated by continuous suction. It healed in about two weeks without noteworthy digestion of the wound. He was dismissed 33 days after admission. At that time a barium meal showed normal emptying of the stomach. Five years later, however, he returned because of complete pyloric obstruction. This was relieved by pyloroplasty. At operation a diverticulum two and one-half inches long and one and one-half inches in diameter arising from the upper surface of the first part of the duodenum was found. We attributed this to the unclosed perforation. It was excised and a modified Finney pyloroplasty performed. The patient has now remained well for two years.

Results.—We have closed 26 duodenal perforations by the method just described. The average age of the patients was 40 years, the limits being 20 and 72 years. All were men. Six patients had drains inserted through incisions in the right flank and suprapubic region. Five of these died. Of the 20 patients whose abdomens were closed without drainage, none died. Two of them had some infection of the wound. One of these developed an incisional hernia.

Twenty-one patients recovered; five died, a mortality of 19.6 per cent. Of 18 patients operated upon sooner than 11 hours after perforation, none died. Of 21 patients operated upon sooner than 24 hours after perforation, only two died, a mortality of 9.5 per cent. Of four patients operated upon 24 hours or longer after perforation, only one recovered. These results, compared with results reported by others who used various operative procedures, demonstrate that the immediate prognosis depends more on the time elapsed since perforation than on the special operative procedure employed. This statement, however, is not true of the late results.

We have been able to trace all 20 of the patients who recovered. Two have died; one of tuberculosis, and one following operation for stone in the common bile duct. This was the only patient who had gallstones. To date, only one of them has required a gastro-enterostomy for duodenal obstruction. The others have remained as nearly free of indigestion as you can with reason expect patients of the ulcer type to remain. Their ulcers have apparently been cured by the perforation. In none of them has duodenal obstruction been caused or increased by the operation, and in all but one, whatever obstruction was present at operation has either disappeared, or has not increased enough to cause symptoms.

CONCLUSIONS

(1) Perforated ulcer of the duodenum can be closed easily and securely by suturing the prepyloric surface of the stomach to the healthy duodenal wall, above, under, and distal to the perforation.

(2) This procedure does not cause, under any conditions, duodenal or pyloric obstruction, and does not increase any obstruction already existing.

(3) Roentgenologic studies show the apparent cure of ulcers so treated, no stasis of stomach contents in the prepyloric region and no delay in their passage through pylorus and duodenum.

(4) None of the five deaths following operation could be attributed to this method of closing the perforation.

(5) The late results of the operation have been excellent. They prove that pyloroplasty or gastro-enterostomy are seldom necessary when it has been used.

(6) The method cannot be used to close perforations complicated by supraduodenal abscess.

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DISCUSSION OF THE PAPERS OF DOCTORS MCGEHEE AND GATCH

DR. WILLARD BARTLETT, JR. (St. Louis, Mo.).—I wish to express my appreciation of the honor of being granted the privilege of the floor and for Doctor McGehee's invitation to discuss his paper.

We have not had in recent years such a case as Doctor McGehee describes in which chronic duodenal obstruction resulted from a developmental anomaly. Perhaps you will find interesting what we have learned from the study of a patient in whom duodenal obstruction of a very high grade resulted from a marginal ulcer subsequent to gastro-enterostomy performed 13 years previously. Obstruction was apparently complete as no bile had appeared in the stool for a number of days before treatment was instituted.

The first objective in the preparation of such a patient for operation should be decompression of the obstructed bowel. The ideal procedure would be continuation of such treatment until edema and spasm have disappeared; these are consequences of the inflammation and infection almost invariably associated with obstruction at any level of the gastro-intestinal tract. The more nearly this ideal can be attained, the more safely and easily can manipulation and suture of the obstructed loops be carried out. Decompression is readily brought about by the employment of continuous suction applied to an indwelling nasal catheter. Since 1930, we have been using a simplified form of the apparatus described by Ward. We prefer it to the Wangensteen apparatus for the latter is merely a siphonage and depends, in practice, upon the accumulation of measurable quantities of fluid in the stomach.

We have reported the plan of measuring the rate of loss of body fluids aspirated from the stomach in ileus and the rate of disappearance from the stomach of ingested fluid as recovery occurs, referring to the former as a negative, and to the latter as a positive, "pyloric balance." The patient under discussion had a negative balance on eight consecutive preoperative days of 4.2, 2.6, 1.7, 2.7, 2.4, 1.5, 1.9, and 2.5 liters per 24 hours, respectively. The immediate decrease in the value of the negative balance, from 4.2 liters to 1.7 liters on the third day of suction, reflected the subsiding of the edema and spasm and the conversion of the complete into an incomplete obstruction and bile reappeared in the stool. The fact that the balance remained persistently negative after this occurred indicated the presence of a mechanical factor in the obstruction and this prediction was borne out at operation by the finding of both scar tissue and torsion as etiologic agents. The large quantity of fluid, even if considered as only water, to be replaced daily is obvious; these amounts must be added to the daily minimum of 1,000 cc. lost as insensible perspiration and the additional 1,000 cc. to which we desire the urine should be diluted.

DR. J. M. T. FINNEY (Baltimore, Md.).—I have been much interested in both of these papers: in Doctor McGehee's for the reason that it brings very vividly to my mind some experiences I had away back 30 or 40 years ago when I met with my first case or two of dilated duodenum. I had not the remotest idea as to what it was, or what the cause of it was, but it did

not seem to me the same cause existed in every case. Some were definitely due to gastromesenteric ileus. You could in such cases demonstrate a definite constriction of the bowel by the vessels, but there were other cases where I could not demonstrate any such obstruction.

The first group were amenable to operation, the operation suggested by Doctor McGehee. I first tried gastro-enterostomy. That helped some but it did not relieve them entirely. The type of case in which I could not demonstrate a definite obstruction was that curious type of thin, enteroptotic individual, long barreled and highly neurotic. It seemed to me an anatomic condition, or possibly functional—whatever that means—a disturbance in the innervation of the bowel wall, because as Doctor McGehee said, one of the things usually found present was an enormously dilated pylorus. The wall of the duodenum was very thin, so one could see through it. That type I gave up as inoperable, closed the abdomen, and turned the patient over to the medical men. My good friend Dr. Thomas R. Brown became interested and he was able to do more for them than I could. I did not know what the condition was, and could not help much with surgery. I had some interesting correspondence at that time with Dr. W. J. Mayo and with the late Dr. A. J. Ochsner. They had both seen the condition. That was 30 or 40 years ago, and at that time neither of them had much to offer.

Doctor Gatch's paper interested me very much. In my experience these lesions are usually on the anterior wall, and if we get them early enough and perform a pyloroplasty, we can excise the ulcer through the incision in the anterior wall of the stomach or duodenum. I have had very satisfactory results with these cases. If for any reason we cannot accomplish this, we must employ some substitute procedure, closing them as best we can. If we are not quite satisfied with the closure, we sometimes take a plug of omentum and suture it carefully over the opening, and then usually put down a small drain to the outside of the plug. One can usually do one or the other of these two things. Doctor Gatch's suggestion, I think, fills the bill beautifully. It seems so obvious that I do not know why some one did not think of it before. It should give a very satisfactory result.

Dr. E. P. Quain (Bismarck, N. D.).—Since you were kind enough to offer me the privilege of the floor, I should like to refer to Doctor Gatch's paper: Some years ago I had the opportunity to become familiar with a method used by certain Scandinavian surgeons in operating for acutely perforated duodenal ulcers. It consists in merely closing the perforation with a suture or two and fastening a tag of omental fat over it and then performing a gastrostomy. This is done by putting a rubber tube into the stomach on the greater curvature about 10 or 15 cm. from the pylorus. The tube is pulled out through a stab wound to the left of the linea alba. This tube will empty the stomach and duodenum and keep them empty and at complete rest while the duodenal wound is healing. I have found this method more easily and quickly done than any of the more radical procedures described, and the postoperative results, both immediate and late, have been very satisfactory.

The subject of chronic duodenal obstruction has interested me for a long time. As some of you may remember, I translated the book Professor Duval wrote on this subject several years ago (Mosby & Co.). There is no question that chronic duodenal obstruction is an entity. In fact, it is several entities. To gain a true conception of this rather common, but complicated, ailment it is necessary to study the whole subject of visceroptosis.

Many obstructed duodena are relieved from operations directed to the cure of right coloptosis, others must have some form of short-circuiting made between the duodenum and the jejunum. But before we get far with these problems we must obtain the cooperation of a well qualified radiologist who has learned to look for other abnormalities in the duodenum than simply that of chronic ulcer. It is necessary also to have an internist, and nurses, properly interested in the subject, because so many of the examinations as well as treatments for these conditions are distinctly medical and not surgical in character. Many patients are relieved and carry on satisfactorily after being taught how to manage their visceroptosis by means of certain postures, binders, pads, *etc.* It is my conviction that our profession has not hitherto paid enough attention to this class of patients. My feeling of satisfaction on seeing some of these lank, lean, visceroptotic and neurasthenic cripples regain their weight and recover their physical and mental health has been one of the finest rewards I have received from the practice of surgery.

Dr. W. O. Bullock (Lexington, Ky.).—I am convinced that there is such a thing as atonic dilatation of the duodenum, for I have had such a case. In this instance the duodenum and pylorus were dilated sufficiently to admit an ordinary sized glass tumbler—there was no obstruction. The wall was thin as Doctor Finney described. I performed a duodenojejunostomy but it did not function. It was easier for the duodenal contents to flow back into the stomach. I then closed the pylorus and performed a gastro-enterostomy which saved the patient's life. (She died six years later of tuberculosis.) I was impressed with the similarity of this condition of the duodenum to that of congenital dilatation of the colon, and believe that the answer to this question lies in the nerve plexus around the celiac axis which is distributed to this portion of the bowel.

Dr. George A. Hendon (Louisville, Ky.).—It stands us all in good stead to have as many expedients as possible, for we often meet with conditions that demand a special type of operation. One which I have had recourse to and which I have found a valuable procedure, particularly in those cases where the edges of the ulcer are cartilaginous and crumbly, and that will not hold the sutures, consists in loosening the upper part of the duodenum as far as the entrance of the common duct and pushing it up into the stomach. This brings four layers of mucous membrane and serous peritoneal surface over the ulcer. When that is accomplished, we perform a gastro-enterostomy to drain the stomach.

Dr. John L. McGehee (closing).—I wish to express my appreciation of the liberal discussion, and especially wish to express my appreciation to Doctor Finney for his observations regarding the subject. The idea in bringing this procedure before the Association was to bring out the fact that there is a type of obstruction of the duodenum, mechanical in nature, of congenital origin, and it is in this type that duodenojejunostomy rather than colopexy is indicated. There is another type in which the duodenojejunostomy does not remedy the situation and the results are disheartening. If the presentation of this case report has brought before you the idea that this condition exists as a clinical entity, and that it can be separated from the other type that is present in the female described by the poet as "limpid sweetness long drawn out," it has been well worth while.

THE SURGICAL ASPECTS OF ACUTE CHOLECYSTITIS

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IT IS with a good deal of hesitation that I venture again to refer to a subject which has been so widely discussed in recent surgical literature. For many years the consensus of surgical opinion favored the conservative treatment of acute cholecystitis; meaning by this term the delay of operative therapy until the acute manifestations of the disease had subsided. An occasional surgeon thought of the advisability of early operation even though he may not consistently have practiced it; and I can recall assisting Halsted, about 1910, in performing a cholecystectomy in the acute stage of acute cholecystitis at a time when cholecystostomy was almost universally the operation practiced. Walton, an English surgeon, in 1923, advocated early or immediate operation in acute cholecystitis, drawing an analogy between the disease and acute appendicitis. But in this country, at least, the propriety of the conservative treatment of acute cholecystitis was not seriously questioned until about 1930. Since that time a number of authors—R. H. Miller,[1] H. F. Graham,[2] Mentzer,[3] Zinninger,[4] Stone and Owings,[5] Judd and Phillips,[6] and others, including myself[7,10]—either have questioned the wisdom of conservative treatment or have opposed it in favor of early operation. The subject is being actively debated in surgical literature, and I was interested to find that in the publications in American literature during the year 1936, writers on the subject are about equally divided, the one group advocating early operation, the other late operation. Some conservatives have become more conservative; for two authors state that if at the time of an emergency operation under a mistaken diagnosis, a simple acute cholecystitis without imminent perforation is encountered, the omentum should be wrapped around the gallbladder, the abdomen closed without drainage and the gallbladder removed at a second operation ten days to three weeks later.

When one studies the literature one is left with the impression that the clarification of certain matters often referred to might aid in determining the comparative value of early and late surgical treatment. I include some of these in the following questions.

(1) Has not the relationship between the clinical symptoms of acute cholecystitis and the pathologic course of the inflammatory process in the gallbladder an important bearing upon the question of early or late operation?

(2) Does the acutely inflamed gallbladder so rarely undergo gangrene and perforation as to disregard these complications as important factors in the treatment of the disease?

(3) Do the complications of gangrene and perforation of the gallbladder contribute so little to the mortality in disease of the gallbladder and bile

ducts that they may be disregarded in a plan of treatment of the disease?

(4) Is the danger to the patient of operating in the acute stage of the disease before gangrene and perforation have occurred greater than the danger of gangrene and perforation, the result of a conservative or waiting policy?

I have made an effort to find an answer to these questions. The material studied includes 150 cases of acute and chronic cholecystitis observed at the Cincinnati General Hospital, 800 cases from the records of the old New York Hospital and 614 cases observed since the opening of the new New York Hospital—a total of 1,565 cases. It includes, in addition, over 35,000 cases of disease of the gallbladder and bile ducts gathered from the more recent literature and some 1,500 cases specifically of acute cholecystitis. It will, of course, be impossible in the time allotted to this paper to report in detail the results of the study of this mass of material. I shall have to state only the conclusions at which I have arrived with respect to the questions I have propounded.

(1) A study of the relationship between the clinical symptoms of acute cholecystitis and the pathologic course of the inflammatory process in the gallbladder shows that there is no direct parallelism between them—in other words, that the clinical symptoms exhibited by the patient do not give any certain indications of the course of the pathologic process in the gallbladder—whether this be toward restitution or toward gangrene and perforation. A careful examination of the clinical symptoms, laboratory findings and pathologic records of 242 cases of acute cholecystitis, which I have personally observed, enables me to make some general statements which seem to hold true. There appears to be a relationship between a high leukocyte count and empyema, gangrene and perforation of the gallbladder and a relationship between the duration of the attack and the severity of the inflammatory process in the gallbladder. It appears that a distinction may be drawn between the cases of acute and subacute cholecystitis on the one hand and the cases of gangrene and gangrene and perforation on the other; and that this distinction lies in the higher temperatures, higher leukocyte counts and higher percentage of palpable gallbladders in the latter groups of cases. But when confronted with individual cases, these observations of a general nature are not very helpful. It remains a fact that in the presence of symptoms, urgent, subsiding or minimal, the pathologic process in the gallbladder not uncommonly proceeds to gangrene and perforation of the gallbladder; and it is often impossible to predicate from the clinical manifestations, the outcome of the disease. This has been the experience of others and is recorded in the literature.

Has not this relationship, or rather, this lack of relationship, a bearing upon the question of early or late operation in acute cholecystitis? In acute appendicitis the impossibility of accurately determining from the symptoms the course of the pathologic process in the appendix is one of the most important reasons for the present attitude toward early operation in this

disease. Since cholecystitis presents similar difficulties in the interpretation of its clinical and pathologic manifestations, it would seem that the same reason for early operation is applicable to, and should be as valid in this disease. Those, however, who favor a waiting policy in acute cholecystitis point out that the two diseases are quite unlike in some respects: that in acute cholecystitis, gangrene and perforation of the gallbladder are infrequent as compared with similar complications in acute appendicitis; and that when gangrene and perforation of the gallbladder occur, perforation into the free peritoneal cavity is far more rare than in acute appendicitis. They point out further that early operation of itself is more dangerous in acute cholecystitis than in acute appendicitis. These matters I shall go on to discuss.

(2) Does the acutely inflamed gallbladder so rarely undergo gangrene and perforation as to disregard these complications as important in the treatment of the disease? In 150 cases of cholecystitis observed at the Cincinnati General Hospital, 80 had acute cholecystitis, and of these, 18 had, at operation, gangrene and perforation of the gallbladder. This number of cases of gangrene and perforation represents 12 per cent for the general series and 20 per cent of the cases of acute cholecystitis. In 800 cases in the records of the old New York Hospital between 1922 and 1932, I found at least 106 cases of acute cholecystitis, of which 16 certainly, and seven additional probably, were cases of perforation of the gallbladder. This number of cases of perforation represents 3 per cent for the general series and 21 per cent of the cases of acute cholecystitis. In 614 cases of cholecystitis admitted to the new New York Hospital, 153 had acute cholecystitis and of these 40 had gangrene of the gallbladder, of which 16 showed definite perforation of the gallbladder. This number of cases of gangrene and perforation represents 6.5 per cent for the general series and 26 per cent of the cases of acute cholecystitis. In the experience of Judd and Phillips the incidence of gangrene and perforation in acute cholecystitis was 13.4 per cent; in that of Zinninger's series, 20.5 per cent; in that of Morris Smith,[8] 22.4 per cent. These and similar statistical data in the literature indicate that gangrene and perforation of the gallbladder occur in approximately 20 per cent of the cases of acute cholecystitis, the pathologic course of which is not interrupted by prompt and appropriate measures.

The statement so commonly made that perforation of the gallbladder occurs only in 2 to 3 per cent in a general series of cases tends to confuse rather than to clarify the situation. It is well known that in a general series, the cases of chronic cholecystitis outnumber the cases of acute cholecystitis, and we are, therefore, simply deceiving ourselves if we base our calculations upon a mixed group of cases, the preponderant number of which are largely outside the problem. When a case of acute appendicitis enters our surgical wards, few of us, I imagine, consider the incidence of gangrene and perforation of the appendix in a general series of cases of appendicitis; rather, we regard acute appendicitis as a condition by itself and weigh the chances of perforation without regard to the chronic form

of the disease. Viewed in the same way—as I think it should be—gangrene and perforation of the gallbladder in acute cholecystitis occur far too commonly to be disregarded as important factors in the treatment of the disease. Let us compare its incidence with that of appendicitis at the New York Hospital. In the first 1,000 consecutive cases of appendicitis operated upon at the New York Hospital since 1932, there were 593 cases of acute appendicitis and 407 cases of chronic appendicitis. In the 593 cases of acute appendicitis, the appendix at operation showed all gradations of acute inflammation but had not perforated in 489 cases, and had perforated with the development of abscess or peritonitis in 104 cases. The incidence of gangrene and perforation in acute appendicitis in this series is, therefore, 17.5 per cent. The incidence of gangrene and perforation in acute cholecystitis observed during the same period is 15.7 per cent, a figure which is reduced to 10.5 per cent if we confine ourselves only to actual perforation with the development of extracholecystic abscess. These figures do not support the contention that perforation of the gallbladder in acute cholecystitis is rare compared to perforation of the appendix in acute appendicitis, although our experience supports the general experience that perforation of the gallbladder into the free peritoneal cavity is uncommon.

(3) Do the complications of gangrene and perforation of the gallbladder contribute so little to the mortality of disease of the gallbladder and bile ducts that they may be disregarded in a plan of treatment of this disease? In the Cincinnati series, the mortality following perforation of the gallbladder was 18.7 per cent; in the old New York Hospital series, it was 34.7 per cent; in the new New York Hospital series it was 12.5 per cent. In the experience of 14 authors with over 500 cases of perforation, the mortality varied between 15 and 65 per cent and averaged 46 per cent. Eliason and McLaughlin,[9] in a study of 555 cases of perforation, found a mortality which varied between 11 and 58 per cent but averaged 47.6 per cent. In a study of 35,000 cases of cholecystitis made to determine the causes of its mortality, *this mortality, i.e.,* the mortality following perforation of the gallbladder, represents 10 per cent of the total mortality following the surgical treatment of noncancerous disease of the gallbladder and bile ducts. This mortality from perforation of the gallbladder in acute cholecystitis is sufficiently high of itself and goes contrary to the idea that it can be disregarded in a plan of treatment of the disease. But I wish to point out that it is not the only mortality which can be charged to gangrene and perforation of the gallbladder. In the study of over 35,000 cases of cholecystitis just referred to, I found that complications following operation, such as peritonitis, hemorrhage, shock, ileus, *etc.*, are responsible for approximately 35 per cent of the total mortality in operations upon the gallbladder and bile ducts; and it is perfectly clear that a part of this mortality is due to operative difficulties and postoperative complications the result of having to deal with the late manifestations of untreated acute cholecystitis such as longstanding extracholecystic abscess, liver abscess, fistulae between

gallbladder and neighboring viscera, adhesions and inflammatory stricture of the common duct.

(4) Is it true, as is commonly stated, that the danger to the patient of operating in the acute stage of the disease is greater than the danger of gangrene and perforation of the gallbladder, the result of a conservative or waiting policy? There is still a fairly widespread opinion that there definitely is great danger to the patient in operating in the acute stage of the disease; a danger greater than that to which he is subjected due to gangrene and perforation of the gallbladder, extracholecystic abscess, peritonitis and other complications the result of conservative treatment. The causes of this presumed or real danger have been stated by some to be the failure of the patient to adjust himself to the acute infection, and the technical difficulties of the operation itself. It is rather difficult from the literature to get at the truth of the matter. The mortality in acute cholecystitis based upon the experience of eight authors in 1,275 cases varies between 4.7 and 22.5 per cent and averages 8.7 per cent; but it is clear that this mortality fails to show what it might have been if a deliberately early attack upon the disease had been made. The mortality in a general series based upon over 35,000 cases varies between 2.6 and 10.4 per cent and averages 6.6 per cent; but this mortality is an inclusive mortality and cannot properly be compared with the mortality in acute cholecystitis. I am able, however, to present a series of 153 consecutive cases of acute cholecystitis in which our policy has been consistently to operate early with the purpose of avoiding the dangers of gangrene and perforation; a policy, the reverse of the conservative policy. The adoption of this policy does not mean that we have operated upon every case as an emergency; for we have deferred operation in those cases in which preoperative treatment seemed necessary.* Nor does it mean that we have succeeded in eliminating gangrene and perforation; for as I have indicated, the gallbladder showed areas of gangrene in 40 of the 153 cases, and in 16 of these it was perforated on admission. But it does mean that the 153 cases were deliberately subjected to operation in the acute stage of the disease and with the hope of avoiding the serious complications referred to. In 138 cases, cholecystectomy was done, in 14, cholecystostomy with drainage and in one, abdominal drainage for generalized peritonitis. In the entire series there were five deaths, a mortality rate of 3.2 per cent.

Analyzed from the viewpoint of the extent of the disease, there were 137 cases of acute cholecystitis without demonstrable perforation of the gallbladder, with three postoperative deaths, a mortality rate of 2.1 per cent. In 16 cases with demonstrable perforation of the gallbladder and with extracholecystic abscess (15) and generalized peritonitis (one), there were two

* Sixty-five per cent of the cases were subjected to operation the day of admission; 35 per cent were observed 48 hours or more before operation was performed. Delay in operation was more often due to difficulties in differentiating between acute cholecystitis and other acute abdominal conditions with which it may be confused, than to the time consumed in getting patients in favorable condition for operation.

deaths, a mortality rate of 12.5 per cent. This mortality rate is actually lower than the average mortality rate in the collected series of 35,000 cases of gallbladder disease quoted above and, with one exception (Judd and Parker:[11] 1,036 cases, mortality 2.6 per cent), is lower than the mortality rate in general series of cases reported from individual clinics. Our experience, while admittedly not large, raises the question whether, after all, the danger of operating in the acute stage of acute cholecystitis is well founded. I think it has been overemphasized and is distinctly a less grave danger than that of gangrene and perforation to which the patient is subjected as a result of conservative treatment.

SUMMARY.—I have found as the result of a study of a large number of cases of acute and chronic cholecystitis that the clinical symptoms, physical signs and laboratory data in acute cholecystitis often fail accurately to indicate the course of the pathologic process in the gallbladder. In the acutely progressive type of the disease, the clinical manifestations fairly closely parallel the pathologic process in the gallbladder; but in other cases, even in the presence of subsiding or minimal symptoms, the pathologic process in the gallbladder may proceed to gangrene and perforation of the organ. Until we have more accurate methods of diagnosis, it would seem proper to take this fact into consideration in our treatment of the disease, just as we do in acute appendicitis; and especially so, if gangrene and perforation occur with any considerable frequency and contribute considerably to its mortality. It appears with respect to gangrene and perforation that these complications occur approximately in 20 per cent of all cases of acute cholecystitis, the pathologic course of which is not interrupted by surgical measures. It appears further that these complications are responsible, under a deferred plan of treatment, for a mortality in acute cholecystitis which varies greatly in the experience of different observers but which rarely in the literature is below 20 per cent and often is as high as 40 per cent; and that this mortality represents approximately 10 per cent of the total mortality in the surgical treatment of noncancerous disease of the gallbladder and bile ducts. This incidence of gangrene and perforation and this mortality are sufficiently high not to be disregarded in a plan of treatment of this disease; unless it be true that they are less a menace to the life of the individual with acute cholecystitis than operation performed early in the disease and with the purpose of avoiding them. That they are not less dangerous but distinctly more dangerous than operation in the acute stage of the disease is suggested by an experience derived from a study of 153 cases of acute cholecystitis in which operation in the acute stage has deliberately been planned and, so far as possible, consistently carried out with the purpose of attempting to lower the mortality from gangrene and perforation of the gallbladder. In this series, the total mortality is 3.2 per cent; but when analyzed from the viewpoint of the extent of the disease, the mortality in 137 cases in which cholecystectomy was performed before perforation had occurred is 2.1 per cent; the mortality in 16 cases subjected to operation after perforation had occurred is 12.5 per cent.

This mortality is so favorable in comparison with the published statistics of mortality rates following the surgical treatment of cholecystitis that I feel justified in continuing a method of treatment which is opposed to the conservative method.

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STRICTURES OF THE COMMON AND HEPATIC DUCTS

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THE subject of strictures of the main bile ducts has been previously discussed in papers from this Clinic, in several papers from other clinics and by individuals particularly interested in the subject. The fact that a great many cholecystectomies are being performed daily, that injuries to the ducts are occurring constantly, that they result in most serious situations and finally that in a considerable experience with this type of lesion we have, in the different cases, employed a variety of procedures to meet varying situations makes it desirable, we believe, to report some of our experiences with these complications, and to describe and illustrate the nine different procedures which we have successively employed in dealing with these strictures.

We have now operated upon 35 patients with stricture of the common or hepatic duct and it seems to me but simple justice to the surgeons of this Clinic to state that in no case did the stricture follow an operation performed in this Clinic.

The causes of stricture of the common and hepatic ducts are quite limited in number. We have seen one case of complete obliteration of the bile duct undoubtedly due to an inflammatory process which completely destroyed the mucosa within the common and hepatic ducts, converting that structure into a fibrous cord without a lumen. Other surgeons have reported such an experience but this fortunately appears to be of quite rare occurrence. Ulceration of the common or hepatic duct or perforation of these structures due to ulceration about a stone occasionally occurs and the formation of a stricture following such an occurrence, as shown in Fig. 1, has occurred in our experience. For practical purposes nearly all of the strictures of the common and hepatic ducts are man made strictures and the result of clamping either the hepatic or common duct in an endeavor to catch and control the cystic or right hepatic artery when torn off in the course of performing a cholecystectomy. This mistake, as shown in Fig. 2, is by far the most common cause of stricture, particularly of the hepatic duct and occasionally of the common duct. If the history of the previous operation in these cases be obtained from the hospital where the original operation was performed, in a majority of the cases of stricture, particularly of the hepatic duct, there will be a description in the operative findings of troublesome cystic artery hemorrhage which was finally controlled by clamps. There will also not infrequently be a history of a prolonged discharge of bile following these operative procedures. In a few other cases, as shown in Fig. 3, an entire section of the common and hepatic ducts will have been removed due to traction upon the cystic duct, angulation or peaking of the common and hepatic ducts so that the right-angled clamp, as shown in Fig. 3, grasps

not the cystic duct but the common and hepatic ducts, severing or ligature of which produces either the removal of a segment of common or hepatic duct or, as shown in Fig. 4, incomplete removal of the anterior wall of the duct at the point where the common and hepatic ducts join. An occasional stricture will result from trauma as illustrated in Case 7, an occasional stricture

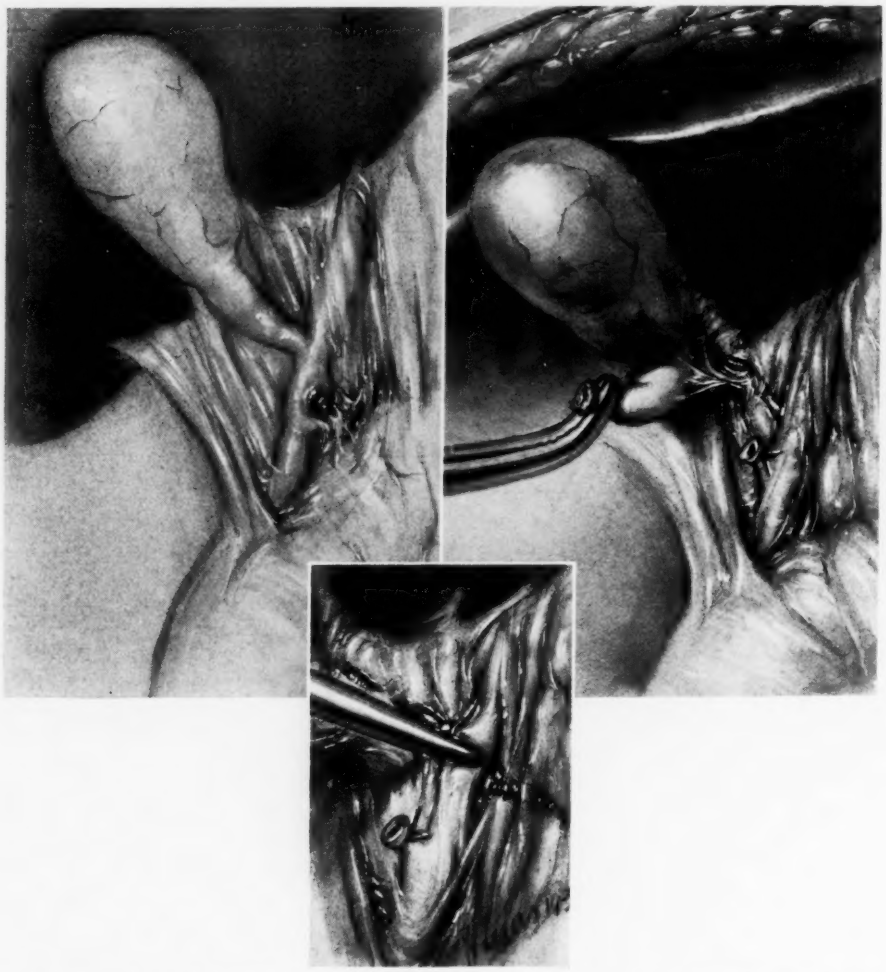

FIG. 1.—The method by which a stone ulcerating through the common duct can produce a stricture.

Insert shows how a clamp which catches the bleeding end of the cystic or right hepatic artery can also catch the hepatic duct and cause stricture.

FIG. 2.—Showing how when the cystic duct is clamped and cut before the cystic artery is clamped and cut, traction is upon the artery and can tear that structure, causing annoying hemorrhage.

low in the common duct following an operative procedure, as in Case 9, will be difficult to explain but is likewise probably due to clamping. There will be, occasionally, patients in whom secondary inflammatory bands may interfere with drainage of the common duct as demonstrated in Case 8.

One should be suspicious of stricture of the common duct in any patient

in whom jaundice appears within a reasonably short time following a previous cholecystectomy unless that cholecystectomy has been performed by the observer and he is quite certain that no injury can possibly have resulted to the common or hepatic duct. When a patient has, in the history of his previous operation, a story of cystic or right hepatic artery hemorrhage controlled by clamping or a history of prolonged drainage of bile following cholecystectomy in connection with technical difficulties during the operation, one should be suspicious that this jaundice is the result of a secondary stricture When in addition this jaundice appears from three, four to five months after cholecystectomy and is painless in character, one should be additionally suspicious that this jaundice is the result of the development of a stricture of the common or hepatic duct.

That strictures of the common or hepatic duct can exist for a number of months or years without the occurrence of marked jaundice seems from our experience quite certain. This is probably due to the fact that the lumen of the strictured duct, while narrowed, is still of sufficient caliber to permit the passage of enough bile as long as that bile is quite normal and of a thin consistency. When, however, secondary infection in the bile passages occurs with thickening of the bile, the appearance of flocculi of mucus together with perhaps an inflammatory reaction within the duct mucosa, this is very probably the factor in these cases which produces late jaundice and obstructive symptoms. When jaundice such as is associated with obstruction of the common or hepatic duct due to stricture occurs, it is painless in character, gradual in onset, late after the previous operation in terms of weeks or months as has already been described, and there is very little that can be done in the way of diagnostic measures to confirm the possible suspicions of a stricture of the common duct. The diagnosis in these lesions must be made largely from the past history and from inference.

A great deal has been written on the treatment of strictures of the common and hepatic ducts. We have published articles on the production of complete external biliary fistulae and the later transplantation of these fistulae into the duodenum, stomach or jejunum. As I have personally stated in the literature and in discussions on this subject, this has for the most part proved an unsatisfactory operative procedure and based upon our experience now with 14 such cases, is an operation which should be reserved as a last resort measure for those patients who have such high strictures at or above the point where the hepatic duct divides into the right and left branches that direct anastomoses between the duodenum or jejunum and the open end of this strictured duct cannot be effected. Based upon our personal experience with this transplantation operation, and because we have written about it, it is my duty, I believe, to state that this operation is far inferior to the direct anastomosis as proposed by Dr. W. J. Mayo, in which the opened end of the hepatic duct is accurately anastomosed to the mucosa and muscularis of the duodenum or jejunum. Unfortunately, direct duct anastomosis will not be possible in a small percentage of cases and in such cases the operation

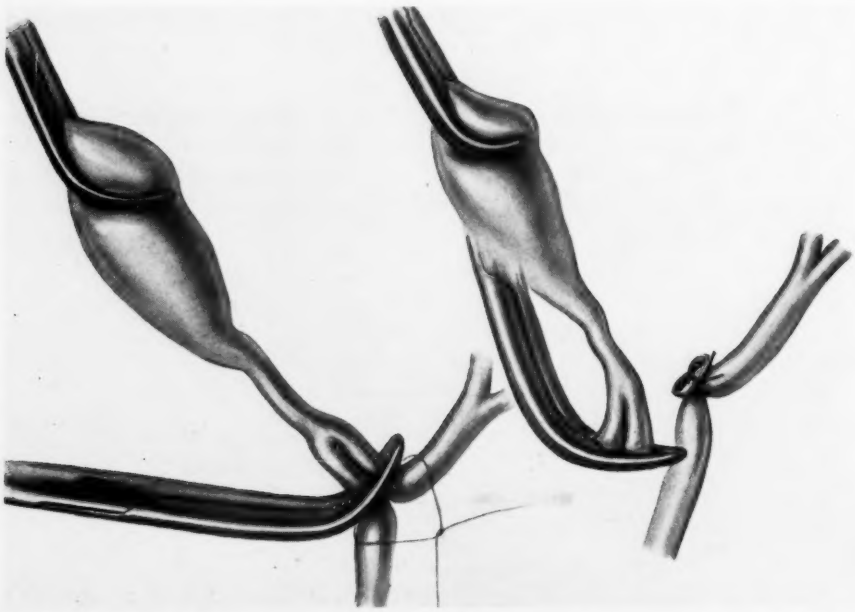

FIG. 3.—Showing semidiagrammatically how an entire section of the common and cystic duct may be removed by applying the clamp too low after pulling up on the cystic duct, as occurred in Case 2.

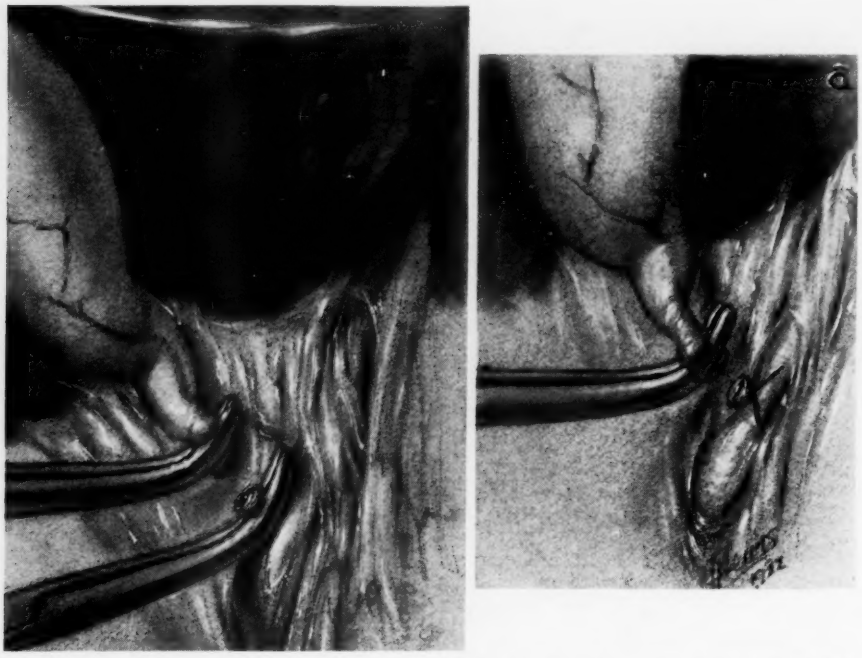

FIG. 4.—Showing semidiagrammatically how stricture of the duct can result from applying the right-angle clamp in cholecystectomy so far down on the cystic duct that it grasps part of the wall of the common and hepatic duct and ligature at this point produces a stricture. Probably the cause of stricture in Case 6.

which we have described in the literature and which was first employed by Dr. Hugh Williams of Boston becomes justifiable in application, since it is then the only procedure possible.

The variety of experiences which we have had with strictures of the common and hepatic ducts can, we believe, best be demonstrated by presenting a few features of nine successfully treated cases, each by a different method, by illustrations of the operative procedures employed in each case and follow up reports as to the end-results.

CASE REPORTS

Case 1.—Mrs. N., age 27. Origin of the stricture, cholecystectomy performed September 9, 1930. Patient made a good recovery but drained bile for eight weeks. Her

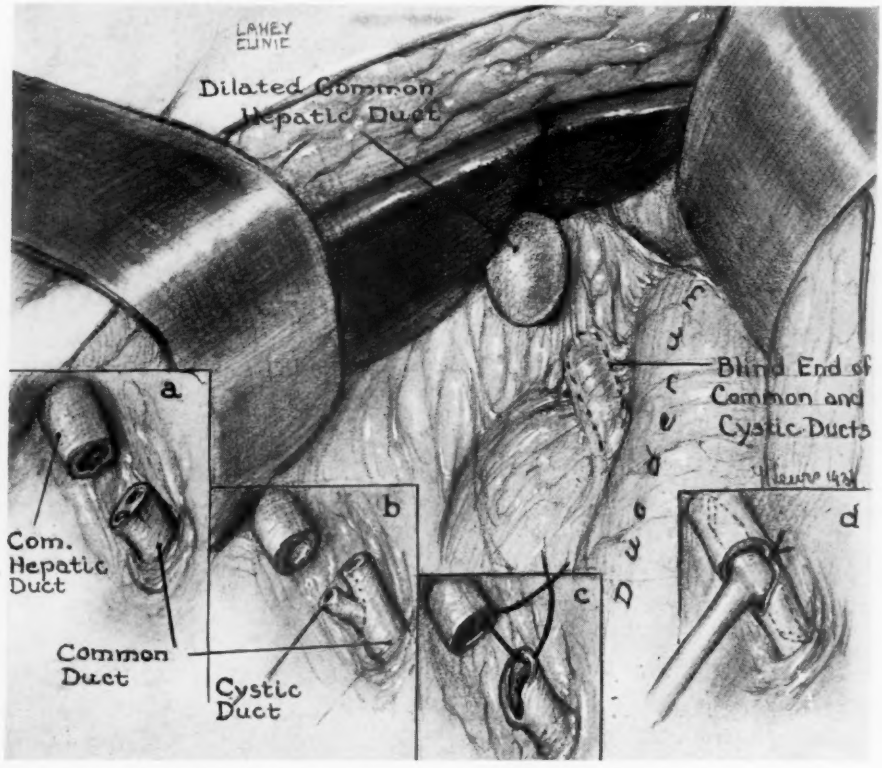

FIG. 5.—End-to-end anastomosis of common duct for complete division of duct following cholecystectomy.

stools were clay colored during this time. Drainage ceased in the last week in November, 1930, and stools became colored at this time. During the first week in December, 1930, the patient became jaundiced and stools clay colored. Jaundice, high colored urine and itching persisted until she entered the Clinic on February 12, 1931 (five months after original operation).

After a period of preparation she was operated upon February 17, 1931. Conditions as presented in Fig. 5 were found. In the main portion of the illustration may be

seen the blind and dilated end of the hepatic duct, and the blind ends of the common and cystic ducts. In Insert a may be seen the end of the common hepatic duct, trimmed off, the end of the common duct, trimmed off, together with the entrance of the cystic duct embedded against the contracted common duct. In Insert b may be seen the stump of the cystic duct dissected free, and in Insert c, the cystic duct incised to serve as a cuff of tissue; also the posterior guide suture bringing together the back wall of the dilated hepatic duct and the enlarged common duct. In Insert d may be seen the T tube inserted, one end in the lumen of the hepatic duct and the other in the lumen of the common duct. The ends of the common and hepatic ducts were brought together about this tube, covered with a layer of omentum and the tube allowed to remain in the common duct for several weeks. Following this the patient had a miscarriage, a subphrenic and a pelvic abscess. The subphrenic and pelvic abscesses were drained. She made an excellent recovery and has had an excellent result. A report in May, 1935, shows no jaundice; patient in excellent condition in every way. A later report, December 3, 1936, states that the patient has never been as well in her life, that she is in her fifth month of pregnancy, uneventful so far, no pain, and no jaundice five years after operation.

FIG. 6.—Complete division of common and hepatic ducts caused by low placing of clamp at time of cholecystectomy.

This case represents a successful end-to-end anastomosis of the severed duct, now of five years' standing with a good result. One of the features making this case successful and remarked upon in the description of the operation is the ease with which the ends of the severed ducts were brought together. When there is any tension due to the defect from loss of the duct itself, stricture will occur and undoubtedly one of the successful features of this case is that there was sufficient slack in the duct to bring the ends together without tension.

Case 2.—Mrs. W., age 25. This patient was operated upon ten months previously after an attack of abdominal pain and vomiting. A diagnosis of acute appendicitis was made and she was operated upon elsewhere. At this time, her appendix was removed

and an empyematous gallbladder, which was greatly dilated, was also removed. On the twelfth postoperative day, she began draining bile, which continued for six weeks and which has persisted, intermittently, since. The sinus opens and closes and when closed the patient feels nauseated and becomes jaundiced. She was admitted to the Clinic ten months after the original operation. She was jaundiced, had lost 12 pounds, her skin was itching, the stools were clay colored and the urine highly colored.

The surgeon referring the case frankly described what he had done at the operation of removal of the gallbladder, as shown in main portion of Fig. 6. A right-angled clamp had been placed sufficiently low so that both the common and hepatic ducts were caught

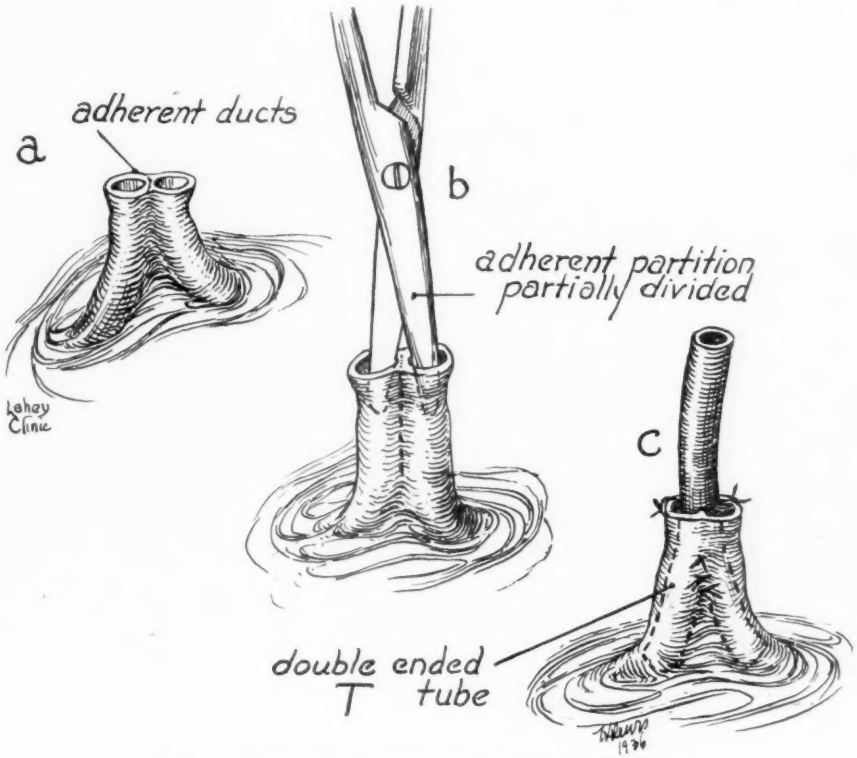

FIG. 7.—Management of common duct stricture seen in Fig. 6.

in a clamp, which were tied and cut across, and when the gallbladder was removed, the gallbladder stump contained not only the cystic duct but a section of the hepatic and common ducts, the hepatic and common ducts being tied together as shown in Insert a.

At operation the end of the hepatic duct was found adherent to the end of the common duct (Fig. 6, Insert a), in the position that a Mikulicz anastomosis would be, both ends of the ducts being blind and closed. The ends of the common and cystic ducts were opened (Fig. 7a). The posterior walls of the two ducts were cut through as in a Mikulicz operation (Fig. 7b), a T tube inserted into the hepatic duct and into the common duct and the ducts sutured about the tube (Fig. 7c). The tube within the ducts was worn from the date of operation, September 11, 1935, until January 4, 1936, at which time the T tube was removed. There has been complete relief. The sinus tract promptly closed following removal of the tube, there has been no jaundice, no pain and no obstruction. Patient gained 25 pounds in weight and was in excellent condition November 25, 1936, 14 months after operation.

The approximation of the ducts by the Mikulicz principle was possible in this case by the fact that the inclusion of both ducts in the ligature at the first operation established fusion of the ducts on their posterior wall and overcame tension.

Case 3.—Mr. A., age 53, was brought to the Clinic by the surgeon who had operated upon him, with the history that on September 5, 1935, while performing a cholecystectomy for acute cholecystitis, there occurred a serious hemorrhage from what appeared to be a branch of the hepatic artery, and in the attempt to control the bleeding point, the hepatic duct had been grasped with a clamp. For 11 weeks postoperatively, this patient drained bile. After the abdominal sinus closed, he developed jaundice for a few days. The wound then broke open and discharged for a few days but again closed shortly without the appearance of jaundice. He had no jaundice until February, 1936, when it returned and has persisted until the present time, June 14, 1936. He has lost 30 pounds in weight. His stools have been clay colored most of the time but occasionally there has been a little bile in them.

At entrance to the Clinic, he was deeply jaundiced. Bilirubin index 8.6. He was operated upon June 19, 1936, five days after entrance. A long, complete stricture of the hepatic duct was demonstrated running up to the bifurcation of the right and left branches of the hepatic duct (Fig. 8a). The distal end of the common duct was smaller than the dilated branches of the hepatic duct, due to the fact that it had not been functioning. The dilated blind stump of the hepatic duct at the point of division was so great that direct anastomosis could not be effected between the ends of the ducts. The hepatic duct stump was so short and imbedded in the liver that an anastomosis between the end of the duct and the duodenum was also impossible.

The dilated hepatic duct at the junction of division of the right and left branches of the hepatic was incised with the escape of a large amount of "white" bile. The undilated portion of the common duct distal to the stricture was opened (Fig. 8b). The scar tissue between the two was incised to make a trough into which a No. 18F. catheter was laid (Fig. 8c and d). One end of this catheter passed down into the common duct but not through the ampulla of Vater into the duodenum. The ampulla of Vater was demonstrated to be patent. The upper end of the catheter rested in the dilated hepatic duct at the point where it divided into a right and left branch. The edges of the scar tissue of the stricture were accurately sutured over this No. 18F. catheter and reinforced with the surrounding peritoneum. The suture was effected so accurately that there seemed no danger of leakage (Fig. 8e). A cigarette drain, however, was placed at the foramen of Winslow and the wound closed.

There was immediate drainage of bile into the duodenum, the bilirubin index three days after operation had dropped to 3.6. The jaundice immediately began to clear and on the twenty-fifth, six days after the operation, dropped to 3.4, and on the twenty-sixth, seven days after the operation, to three milligrams, on the twenty-eighth, to 2.6, on July 2, to 2.5, and on July 6, 17 days after operation, to 2.1. This patient made an uneventful recovery and never had any subsequent drainage of bile. The cigarette drain was removed on the eighth day. The wound closed firmly. He has gained 25 pounds in weight, and a letter from his physician, December 1, 1936, states that he had no difficulties whatever.

This case presented a difficult problem. The stricture was so high, running as it did up to the division of the hepatic into its right and left branches, and so deep in the bed of the liver that an anastomosis between the opened end of the hepatic duct and the duodenum or jejunum would have been very dangerous, if not impossible. The length of the stricture was so great

that end-to-end anastomosis or any plastic operation upon the duct was out of the question. Our experience with the production of external biliary fistulae and their transplantation has been so unsatisfactory that it did not

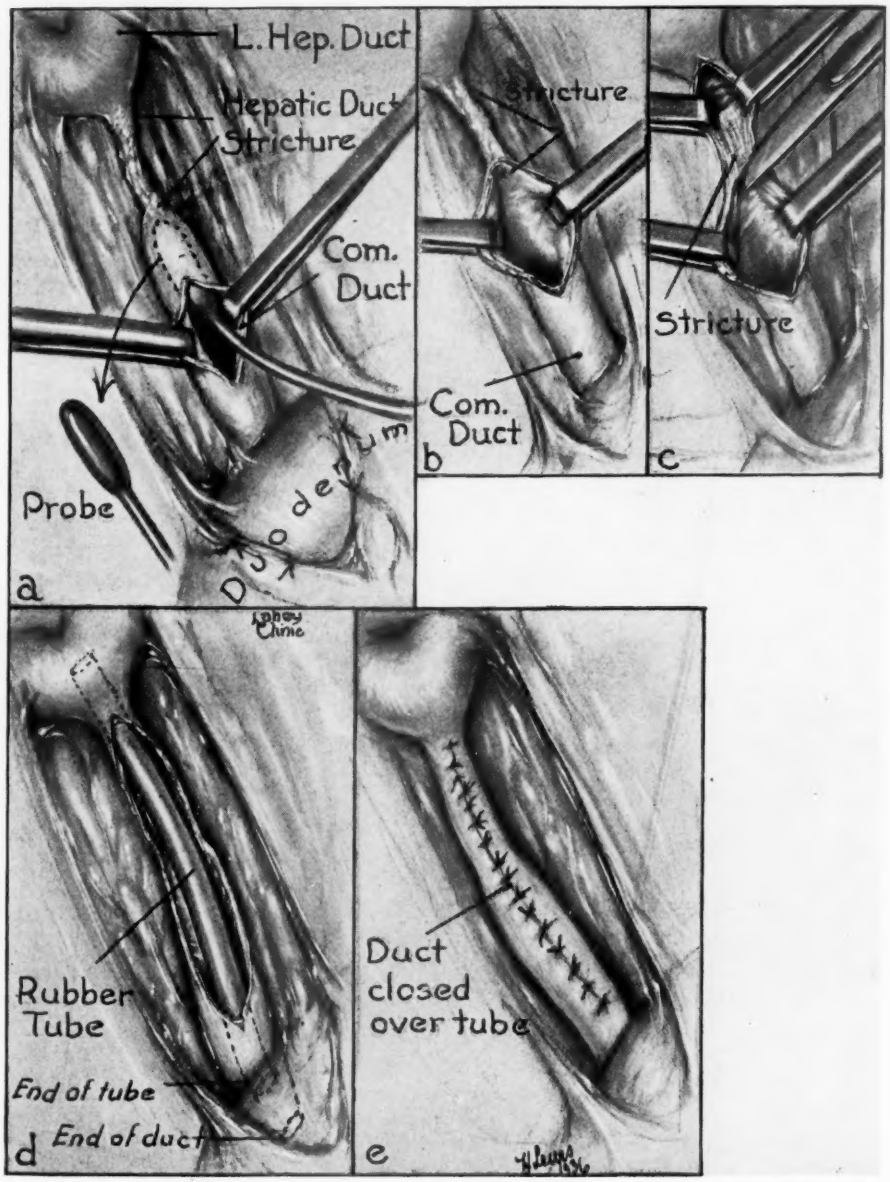

FIG. 8.—Stricture of hepatic duct caused at operation by clamping of the duct in an attempt to control hemorrhage from hepatic artery. Illustrations demonstrate method of treatment of this stricture.

seem worth considering. It must be admitted that the introduction of a rubber tube into the common duct to replace the duct is in some measure a makeshift procedure, but in this patient there seemed nothing else to do and

so far the results have been most gratifying and satisfactory. It is of interest to note that we have removed a tube from a common duct which had functioned well for seven years and replaced it with another still functioning satisfactorily. It will be of interest to observe this case and should he have difficulty with this tube eventually, I feel sure that there will be no difficulty in replacing it. On the one hand, we had a patient deeply jaundiced with no method of reconstructing the duct with his own tissue, and on the other, the uncertainty associated with the introduction of a foreign body. So far, now six and one-half months after the introduction of the tube, it has proved a completely satisfactory substitute for the destroyed duct.

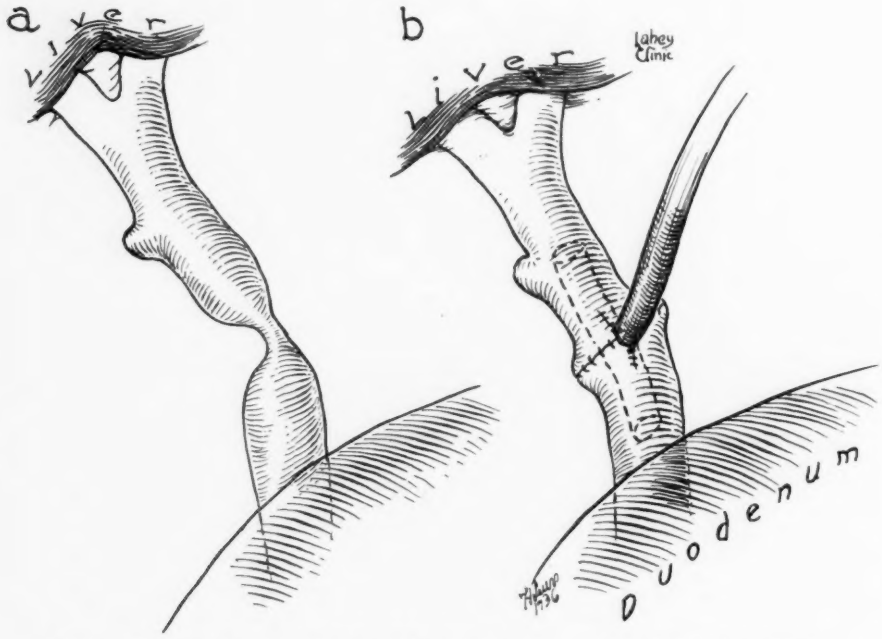

FIG. 9.—Stricture of common duct caused by operative trauma and method of management.

Case 4.—Father S., age 59, was operated upon in 1931 for abdominal pain, jaundice, nausea and vomiting. The operative report states that the patient was obese and the operation difficult. He had symptoms of bile duct obstruction immediately after the operation while still in the hospital, was repeatedly jaundiced, and since the operation, up to the time of his entrance to the Clinic, February 6, 1936, he has had almost weekly attacks of pain and jaundice. At the time of entrance he was jaundiced and was admitted with the diagnosis of probable common duct stricture with or without stones.

Operation was performed February 10, 1936. The dense adhesions from the previous operation were separated from the bed of the liver and the gastrohepatic ligaments isolated. The dilated common duct, at a point just below where the cystic duct enters the junction of the common and hepatic, was found, opened, and several gallstones were removed. The bile in this dilated common duct was thick and definitely infected. The right and left hepatic ducts were explored and all stones removed from them. It was then found impossible to pass a probe downward into the lower portion of the common duct. A search was made for a small fistulous opening but none could be identified.

The common duct was then found distal to this apparently blind end of the common duct; it was isolated and opened. It was dilated to about the size of a man's thumb. When this portion of the duct was opened, a fistulous connection between the two segments of the duct was found which measured approximately two millimeters in diameter. The stricture in this portion was very short, representing practically a diaphragm between the two ends of the common duct. The anterior portion of this diaphragm or stricture was incised, a large T tube was placed in the strictured portion with one arm going into the hepatic and one into the common duct (Fig. 9). The common duct was reconstructed about this so that the reconstructed portion was of greater caliber than the rest of the duct. The lower end of the common duct was investigated to make sure that the ampulla of Vater was patent and dilators readily passed through it into the duodenum. A small cigarette drain was placed down to the foramen of Winslow and the wound closed.

This patient made a very satisfactory recovery, returned home with the T tube in place for four months. The jaundice promptly cleared up. The T tube was removed without difficulty at the end of four months. Bile drained freely into the intestinal tract and all external drainage ceased following removal of the T tube. The last report, December 2, 1936, ten months after the operation, is that this man is in excellent condition with no further jaundice.

The fact that this patient had a very narrow stricture amounting almost to a diaphragm, the fact that it could be reconstructed with no tension whatever on the duct, offers, we believe, excellent prospect of a good result in this case.

Case 5.—Mrs. K., age 42, had a cholecystectomy performed in March, 1934, about one year before admission to the Clinic. Following the removal of her gallbladder she drained bile for six weeks. She was in relatively good health after convalescence from her cholecystectomy until December, 1934, about nine months after operation. On January 1, 1935, she became jaundiced, her urine dark, and stools light colored, and she began to lose weight. The jaundice had steadily, progressively and plainlessly increased. Her stools, however, contained a small amount of bile. The report of her first operation states that after the gallbladder was removed, it was found so dilated that there was no cystic duct, that the common and hepatic ducts ran directly from the gallbladder and both had been cut. At the time of operation, a T tube was introduced into both ends of cut ducts which were then brought together over the T tube. This patient was admitted to the Clinic March 11, 1935, one year after her cholecystectomy, with a diagnosis of stricture of the common or hepatic duct. She showed plus four jaundice, was dehydrated, and suffered from severe pruritus. She had lost 30 pounds in weight. Bilirubin index 11.6.

Operation, March 15, 1935. The gastrohepatic omentum was freed of the adhesions resulting from the previous operation with difficulty, and the dissection was carried down to the region of the common and hepatic ducts. The proximal stump of the hepatic duct was found dilated and about the size of an adult's thumb, projecting out from the bed of the liver for a distance of about two centimeters (Fig. 10). The distal end of the common duct was not seen. An old, partly obliterated fistula seen along the usual course of the common duct was demonstrated and was not opened because of the dense scar tissue and because it would have been impossible to have reconstructed the duct. It was decided to anastomose the dilated end of the hepatic duct to the duodenum. An accurate anastomosis was effected between the stump of the hepatic duct and the duodenum (Fig. 10), a double layer of interrupted sutures accurately approximating the mucous membrane of the duodenum to that of the duct.

The operative recovery was uneventful, there was no discharge of bile, the stools became promptly colored and have remained so since. The bilirubin index on the

so far the results have been most gratifying and satisfactory. It is of interest to note that we have removed a tube from a common duct which had functioned well for seven years and replaced it with another still functioning satisfactorily. It will be of interest to observe this case and should he have difficulty with this tube eventually, I feel sure that there will be no difficulty in replacing it. On the one hand, we had a patient deeply jaundiced with no method of reconstructing the duct with his own tissue, and on the other, the uncertainty associated with the introduction of a foreign body. So far, now six and one-half months after the introduction of the tube, it has proved a completely satisfactory substitute for the destroyed duct.

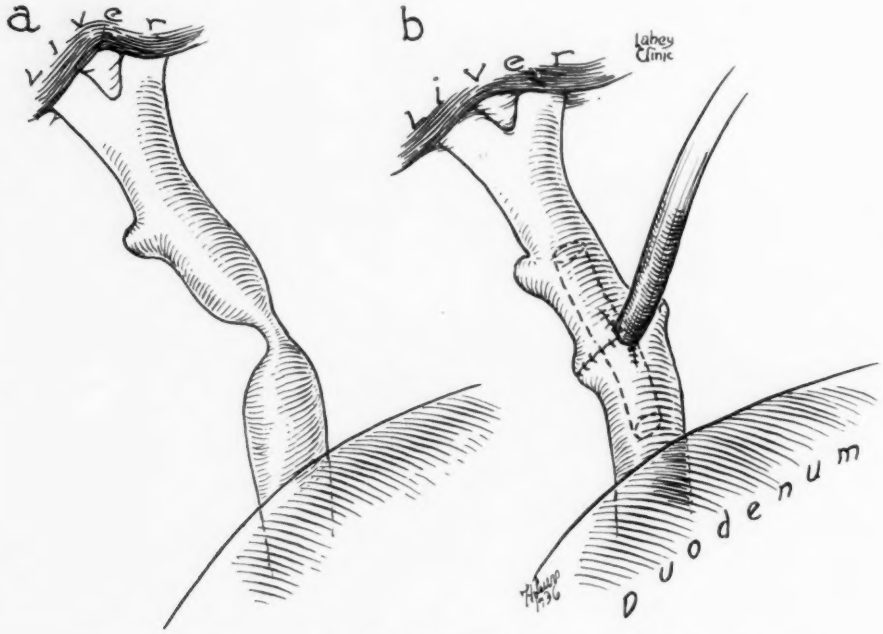

FIG. 9.—Stricture of common duct caused by operative trauma and method of management.

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The operative recovery was uneventful, there was no discharge of bile, the stools became promptly colored and have remained so since. The bilirubin index on the

eighteenth, three days after operation, had dropped to 8.9, on the twenty-first, six days after operation, to 4.1 and fell progressively to normal. An uneventful recovery ensued. A report, December 5, 1936, states that she is and has been perfectly well, and represents the typical and satisfactory case, in which it is possible to make a direct anastomosis between the cut end of the duct and the duodenum.

Case 6.—Miss F., age 28, entered the Clinic March 19, 1936. She had had a cholecystectomy 18 months previously, in July, 1935. Her physician reports that at the time her cholecystectomy was performed the common and hepatic ducts were injured and repaired. Following this repair, the patient drained bile for three months, after which time the sinus closed. Following closing of the sinus, the patient was well for one month, when she became jaundiced, which has since become progressively deeper.

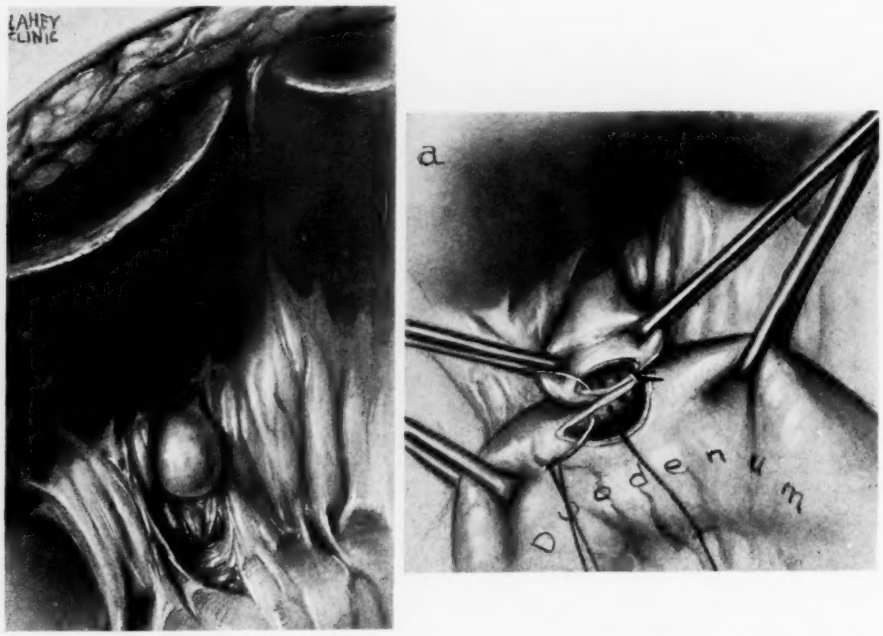

FIG. 10.—Anastomosis of stump of cystic duct to duodenum for complete division of common duct.

She has lost 50 pounds in weight since her operation. On admission she was deeply jaundiced with an enlarged liver, and an incisional hernia. Bilirubin index 10.1. She was admitted with a diagnosis of stricture of the common duct.

Operation, March 23, 1936. The dense scar tissue resulting from the previous operation was dissected away from the liver, the gastrohepatic ligament demonstrated, and the duodenum found to be drawn tightly against the liver at the base of the gallbladder fossa. After dissecting the duodenum away, the dilated hepatic duct was found markedly distended with bile, and the common hepatic duct was dissected free and isolated. The common hepatic duct was opened and noted to contain clear hepatic bile (Fig. 11). A definite stricture of very short length was found to exist between the hepatic and the common ducts, which was incised longitudinally. The lower limb of a size No. 18F. T tube was passed through it and into the duodenum. This tube was passed through the incised stricture by way of an incision in the hepatic duct above the level of the stricture. The upper limb of the T tube was cut short so that it would reach into the right hepatic duct. The strictured portion of the duct was reconstructed after the Heineke-Mikulicz

plan over the lower limb of the T tube in order to widen the lumen of the duct at the expense of its length. This allowed the duct to be well closed, to be of greater caliber than the duct itself and the suture line was under no tension whatever. A cigarette drain was placed down to the foramen of Winslow, it having been previously demonstrated that the sphincter of Oddi was patent; the wound was closed.

The bilirubin index on March 27, four days after operation, had dropped from 10.1, its height at entrance, to 4.9. Three days later, on the thirtieth, to 3.6, and from then on it gradually fell to normal. This patient has made an excellent recovery and at the last report, December 1, 1936, was in excellent condition.

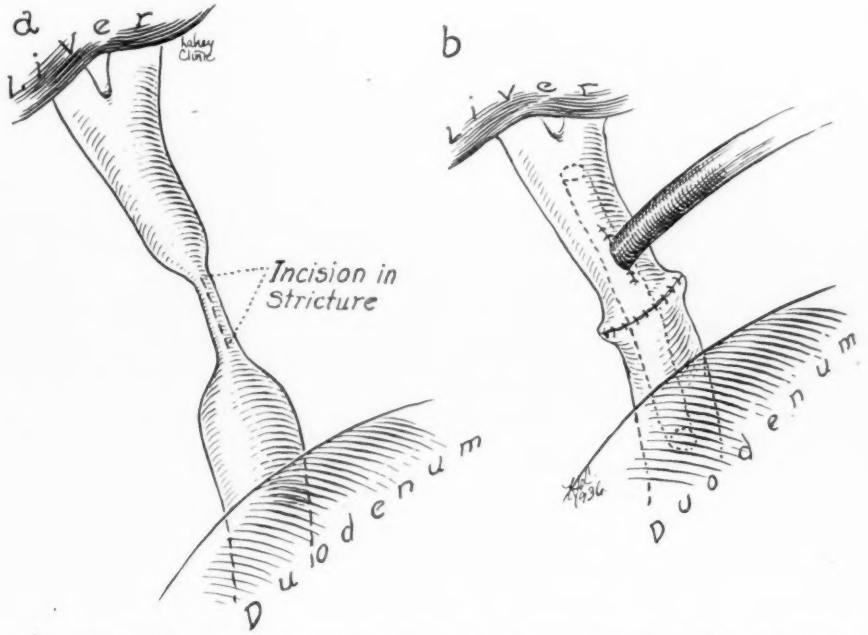

FIG. 11.—Longitudinal incision of common duct stricture and closure about "T" tube. Note position of upright limb of "T" tube in relation to line of closure of stricture.

This case represents a method of closing the duct over a T tube with the upright limb of the tube emerging through the unstrictured portion of the duct and the duct completely sutured about the lower end of the T tube. This, when possible, is much superior to the suture of the duct about a tube which emerges through the center of the suture line as shown in Fig. 9, since the removal of the tube may very well break down the suture line and the defect left by its removal result in a stricture.

Case 7.—Mrs. M., age 27, entered the Clinic September 20, 1930. She had been in an automobile accident July 26, 1930, remaining in a hospital for three weeks, where she was told that she had intra-abdominal injuries. She had a good deal of vomiting but soon after returning home noticed that her eyes were becoming yellow. She was admitted with a diagnosis of questionable catarrhal jaundice. Duodenal drainage was instituted September 24. On stimulation, 50 cc. of turbid yellow material were obtained which contained considerable mucus and pus and tall columnar epithelial cells. The gallbladder did not empty and no conclusion could be drawn. She was discharged from the hospital three weeks later and returned to the Clinic two months later, the jaundice having persisted and having become deeper. She suffered from severe pruritus, the stools were a

very light brown, and her urine very dark. In view of the fact that this jaundice had persisted for four months and had become more intense, it did not seem likely that her condition was due to catarrhal jaundice; on the other hand, she had no pain, or evidence of stones or malignancy.

Operation, February 14, 1931. On exploration her gallbladder was found slightly enlarged and distended, with thick walls. A cicatricial area was found just above the duodenum over the common duct, and a large soft area was noted in the liver substance filled with necrotic pale brown material (Fig. 12). This was evacuated. It was apparently an old rupture of the liver in which healing had not completely taken place. The scar tissue in the neighborhood of the duodenum completely occluded the common duct. The gallbladder was opened and found to contain dark, green bile. The hepatic duct was opened and the right and left hepatic ducts were probed and found to be patent, but the duodenum could not be entered because of the scar tissue resulting from the previous trauma. A T tube was inserted in the right hepatic duct and a large drain was inserted into the gall bladder (Fig. 13). These drains were inserted for the purpose of relieving the existing jaundice and the anticipated relief of stasis resulting after the contemplated anastomosis of the gallbladder to the stomach, duodenum or jejunum at a later date.

Her bilirubin index at entrance was 10.7. On February 26, 12 days after operation, it was 6.3, and on March 2, 18 days after operation, it was 5.4. Following this, with further drainage from the gallbladder and hepatic duct, the bilirubin index soon became normal. The catheter was withdrawn from her hepatic duct 12 days after operation and she continued to drain bile well from her gallbladder. The patient was sent home draining all of her bile from her gallbladder sinus, to return for secondary anastomosis of the gallbladder to the stomach or duodenum.

On April 27, two and one-half months after the original drainage operation, since all bile was being discharged through the gallbladder fistula, the patient was reoperated and the fundus of her gallbladder anastomosed to the stomach. She made an excellent recovery. A report, November 27, 1936, states that she is in good health, with no difficulties.

This patient represents a traumatic stricture of the common duct, drainage of the gallbladder and hepatic duct for relief of jaundice, and successful secondary anastomosis of the gallbladder to the stomach.

Case 8.—Miss B., age 40, entered the Clinic May 19, 1936, because of nausea and abdominal pain. She had had a cholecystectomy in July, 1935. This is said to have been performed for nausea, vomiting, and upper abdominal pain. One stone was found in the gallbladder. She felt well for two or three weeks following operation, when she had another attack of pain and vomiting very similar to the first one, and these have recurred at frequent intervals since that date. Jaundice has not been noted since the operation. She was admitted with a diagnosis of possible common duct stones. Bilirubin index normal, there were no evidences of jaundice, and her stools were well colored. Duodenal drainage was attempted, but because of pylorospasm, could not be satisfactorily conducted. In the course of the attempted duodenal drainage the patient had attacks of pain characteristic of those of which she had previously complained, consisting of a severe twisting and grinding epigastric pain, radiating through into the back, but not referred to the shoulder. It was colicky but did not subside completely between each wave of pain. It required morphine to relieve it. Roentgenologic examination of the stomach showed a somewhat distorted, sensitive duodenum with evidence of adhesions in the second portion, producing sharp angulation. The note from the Gastro-Enterologic Department stated that since the patient has never been jaundiced either before or after operation a year ago, the character of the pain and the failure to find stones in the common duct at the time of her previous operation make it unlikely that she has a com-

Fig. 12.—Stricture of common duct following auto accident with intra-abdominal injuries.

Fig. 13.—Drainage of hepatic duct and gallbladder preliminary to cholecystgastrostomy.

mon duct stone. The probable diagnoses are: Duodenal adhesions; recurrent subacute pancreatitis.

Operation, May 25, 1936. The duodenum was found drawn upward beneath the liver, adherent to that structure at the point where the gallbladder had been removed, covering the lower portion of the gastrohepatic ligament. On freeing the duodenum and turning it down from its abnormally high position, a widely dilated common duct was seen. This duct was covered anteriorly by the right hepatic artery which made an abnormally sharp curve to the right. About one and one-half inches above the point where the common duct becomes retroduodenal, a firm adhesive band passed between the right hepatic artery, across the common duct in such a position as to produce a very marked constriction and marked dilatation of the common duct above this point. This band so tightly constricted the duct that it gave the impression that a string had been tied about the duct at a point just below what appeared to be the ballooned-out portion of the duct above the obstructed region. The distal end of the common duct at a point below the constriction was the size of a normal common duct (Fig. 14a). On cutting this adhesive band with scissors, the duct at the point of obstruction at once returned to its normal size. It was opened and explored (Fig. 14b). No stones or further strictures were found. A No. 7 Bakes' dilator passed easily into the duodenum. A T tube was placed in the common duct and a cigarette drain to the foramen of Winslow. An uneventful recovery followed and a report, July 24, 1936, showed no gastro-intestinal symptoms, or any trouble whatsoever. A later letter, December 3, 1936, reports that this patient has had no return of the pain since operation, no indigestion or nausea and has gained 12 pounds in weight.

This case represents cicatricial obstruction of the common duct by a band following a previous cholecystectomy, severing of which resulted in complete relief. It is of interest to realize that such bands can exist and that, as shown in Fig 14, marked dilatation of the duct can result from these, and symptoms of incomplete obstruction can result.

Case 9.—Mrs. K., age 55, had had a cholecystectomy in January, 1935. At that time her bile duct was opened but no stones were found. She had previously had two severe attacks of pain accompanied by slight jaundice. Three days after operation, she became deeply jaundiced, nauseated, and vomited. The jaundice persisted for six or seven weeks causing intense pruritus. In October, 1935, she entered the Mercy Hospital in Pittsburgh where at a second operation the common duct was reported to have been found as large as a segment of intestine and a stricture was found at about the point where the common duct passes behind the duodenum. A T tube was inserted in the common duct for the relief of the jaundice which cleared up after about two months. Following removal of the T tube a complete external biliary fistula persisted up to January, 1936, at which time the discharge of bile ceased and the stools showed a little color. The fistula started draining again after three days and has persisted to the present time.

She entered the Clinic March 20, 1936, with a Grade II icterus. All bile was discharging from the biliary fistula and the stools were clay colored. Bilirubin index 1.7. A diagnosis of stricture of the common duct was made.

Operation, March 24, 1936. The external biliary sinus was carefully dissected down to its point of origin at the common duct midway between the point where the cystic duct enters the common duct and where the common duct becomes retroperitoneal. This fistulous tract was well nourished, was not attached to the bed of the liver and could be separated and kept intact. The common duct was then carefully separated from its numerous adhesions and found to be markedly dilated. The stump of the cystic duct was found adherent to the portal vein behind the common duct; it was isolated and carefully freed. This duct was about the size of one's little finger and about one inch in

length. The junction between the cystic duct and dilated common duct was carefully dissected and identified. There was no evidence of stones in the common duct, but a definite fibrous stricture just above the duodenum was demonstrated (Fig. 15). It was

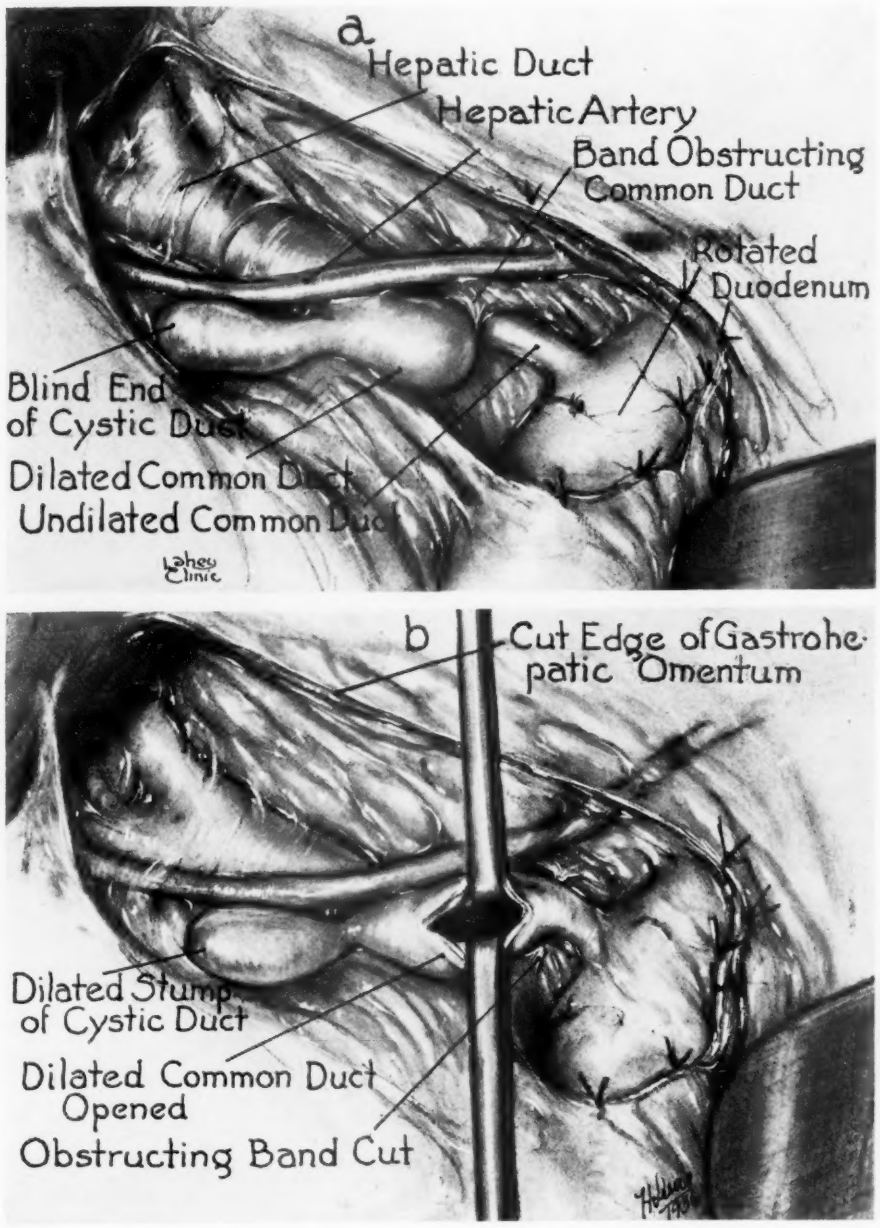

FIG. 14.—(a) Common duct stricture caused by adhesive band of scar tissue. (b) Release of adhesion and exploration of common duct.

thought advisable to transplant the dilated cystic duct into the first portion of the duodenum and this was effected without difficulty. The end of the dilated cystic duct

was cut off, a small section of rubber catheter was tied into the opened end of the cystic duct so that it would not be strictured by the purse string which is placed in the duodenum when implanted (Fig. 16a). It was implanted into the duodenum by two guide sutures and anastomosed to the duodenum by a circular purse string suture (Fig. 16b).

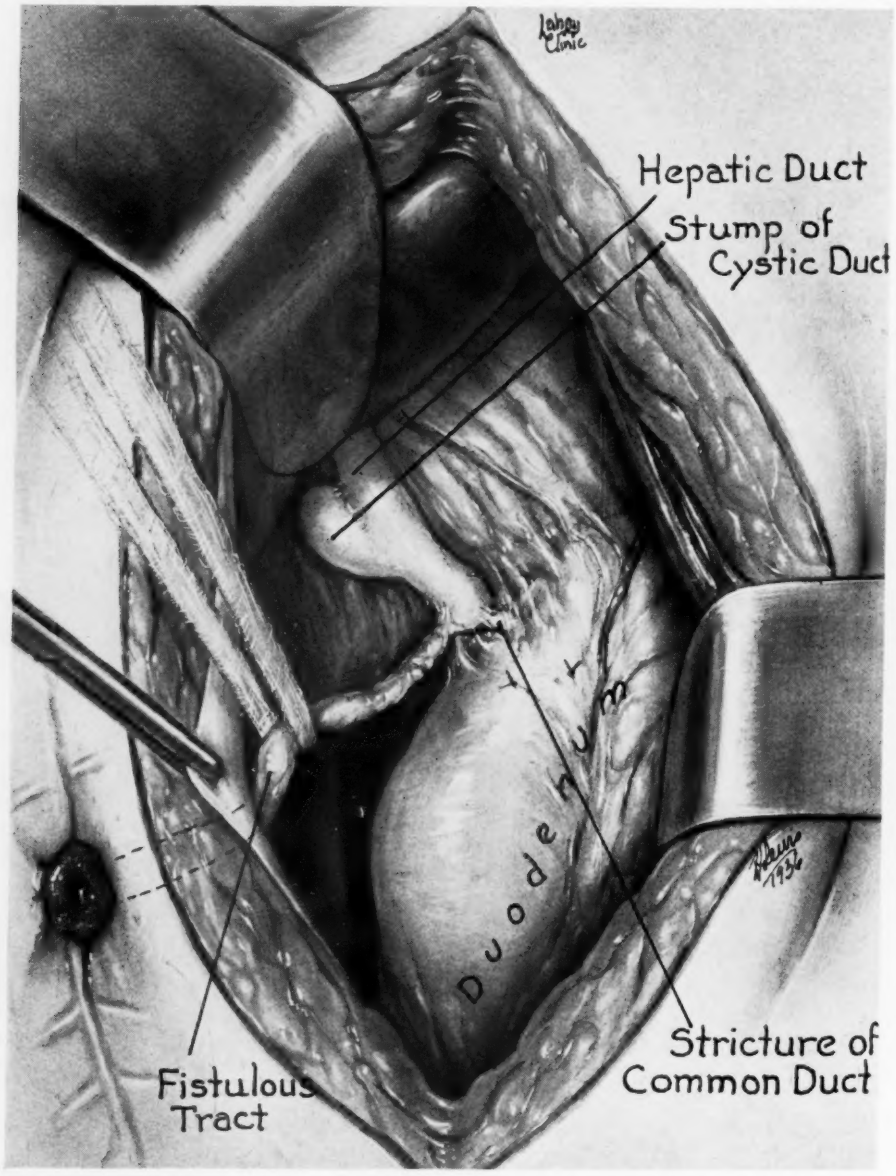

FIG. 15.—Stricture of common duct caused by adhesions just above the duodenum and fistulous tract of external biliary sinus.

One of the problems of the operative procedure was the disposition of the biliary fistula. If this were left in the wound, it would discharge bile and infect the wound. It therefore seemed wise to implant the end of this fistula into the stomach in order to care for its

drainage. Two openings were made in the stomach beyond the pylorus (Fig. 17a). A hemostat was passed through the opening nearest the cardiac end of the stomach and out of the opening near the pyloric end of the stomach. The fistulous tract was then pulled into the stomach, the incision near the pylorus closed about it and the incision in the stomach toward the cardiac end closed (Figs. 16b and 17a and b). This resulted in a discharge of all the bile from the common and hepatic ducts through the cystic duct, implanted into the duodenum, and a discharge of any bile along the fistulous tract into the stomach where it would not infect the wound. Following completion

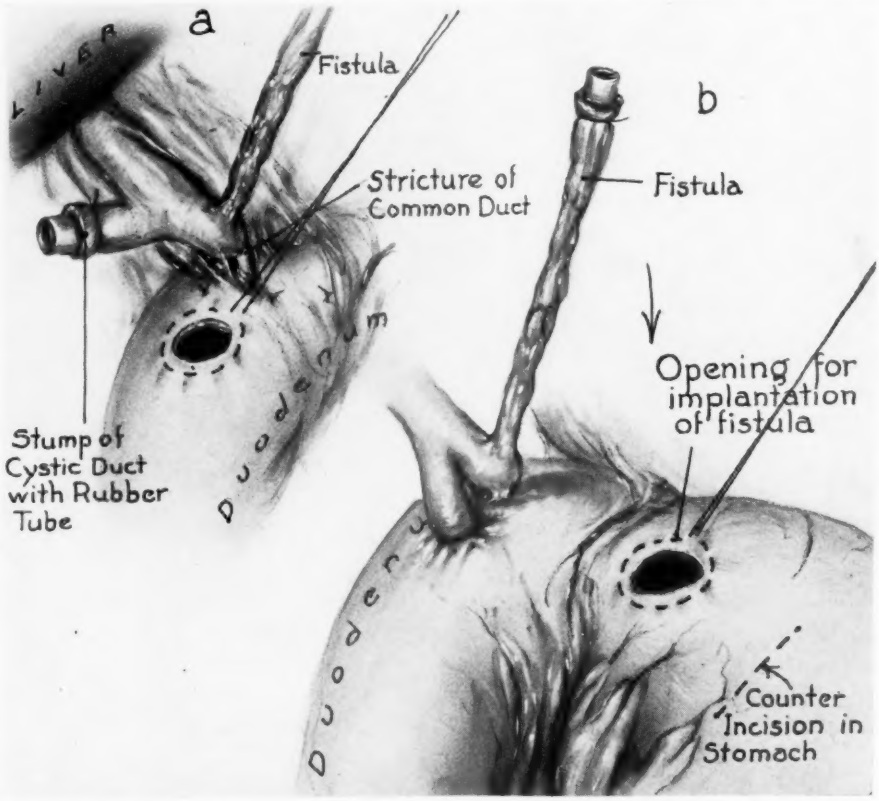

FIG. 16.—Implantation of stump of cystic duct into duodenum and opening in stomach for implantation of external biliary sinus.

of this procedure, it seemed probable that the implantation of the fistula near the pylorus, plus the implantation of the cystic duct into the duodenum near the pylorus, might result in some interference with pyloric function. Gastro-enterostomy was then performed without difficulty and the wound closed (Fig. 17c). This patient made an excellent recovery and had no postoperative difficulties whatever. Her bilirubin which rose for a time after operation soon became normal and she is now entirely free from symptoms. A report, November 28, 1936, states that she is, and has been, perfectly well with no jaundice and no symptoms.

This case represents the rather complicated problem of rerouting the bile from the obstructed common duct (probably seriously injured when opened at the original operation, and not the result of the procedure at the

second operation) into the duodenum and at the same time arranging for the disposal of the discharge of any bile from the tract of the external biliary fistula. This problem was very satisfactorily managed by implantation of the end of the dilated stump of the cystic duct into the duodenum and by implantation of the fistulous tract into the stomach. The gastro-enterostomy, while possibly unnecessary, had to be performed as a precaution against pyloric obstruction, since, had the pyloric obstruction eventuated postoperatively, the secondary performance of a gastro-enterostomy would have been

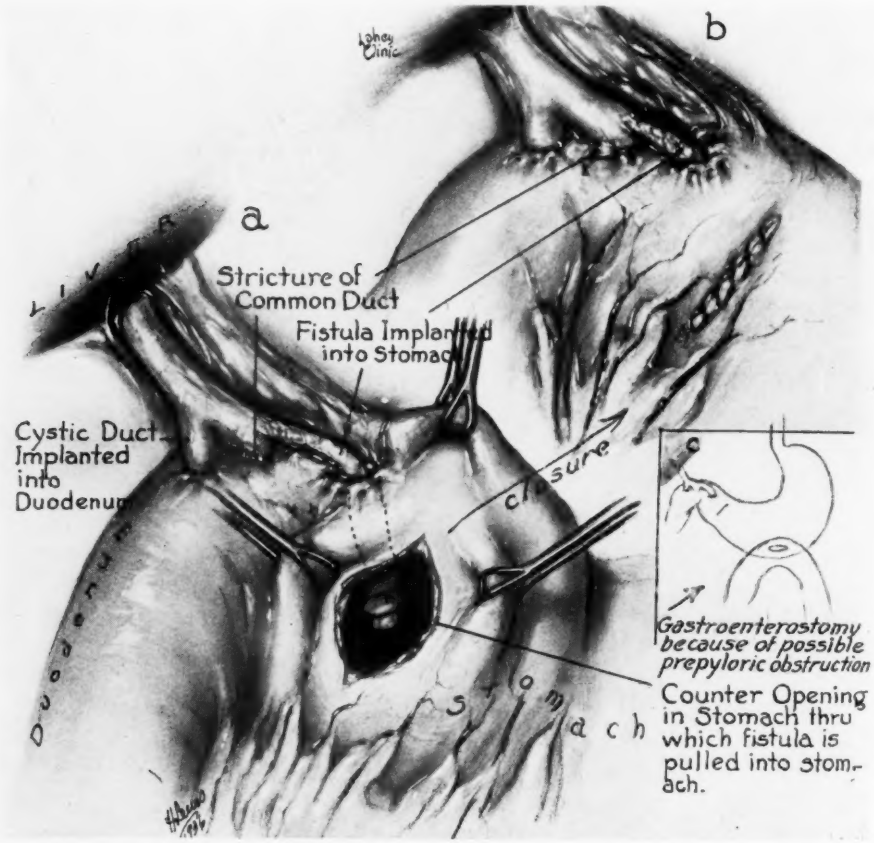

FIG. 17.—Implantation of external biliary sinus into stomach followed by gastro-enterostomy.

quite impossible and the operation quite probably would have resulted in a fatality.

SUMMARY.—Nine cases of stricture of the common or hepatic duct are presented. These cases were chosen from a group of such complications because each one was treated by a different plan, selected to meet what appeared to us in each of these cases to be a different problem and also because these procedures have all been, at least so far, successful in overcoming the difficulties.

Based upon a considerable experience with hepatic duct strictures and also a considerable number of failures as well as successes, I would like to submit a few conclusions which I have drawn from our failures as well as our successes.

CONCLUSIONS

In operating upon common and hepatic duct strictures one should attempt to select the operative procedure which offers the best chance of draining the bile into the intestinal tract at the first operation, as secondary operations after failures upon patients with these lesions are much more complicated and much less likely to be successful.

Repairs fashioned after the Heineke-Mikulicz plan in long strictures of the common or hepatic ducts are doomed to failure since there will be too much tension on the sutures in the reconstructed duct. In such cases, anastomosis of the end of the hepatic duct with the duodenum will be a much more satisfactory procedure.

If a strictured duct is to be repaired by the Heineke-Mikulicz plan of plastic upon it, the T tube is best inserted through a separate incision in the duct above the stricture, the lower limb of the tube passing through the incised stricture as in Case 6 and the plastic performed over this portion of the tube. This permits withdrawal of the T tube without injury to the line of the anastomosis. End-to-end anastomosis of the common and hepatic ducts will be followed by separation unless there is a good deal of slack in both ends of the ducts and they come together easily. If they do not, it is much better to anastomose the end of the hepatic duct with the duodenum.

When the hepatic duct has been cut previously, careful inspection should be made of the amount of hepatic duct available for suture to the duodenum and a decision made as to whether or not a sufficient amount of duct projects from the liver bed so that a safe anastomosis can be effected. This is, in our hands, made easier by the employment of interrupted sutures.

When a rubber tube must be implanted as a substitute for a destroyed duct so that it projects into the duodenum, it is less likely to be successful than when it can rest in the hepatic duct and common duct to bridge a defect and not pass to or through the sphincter of Oddi.

The implantation of the end of a cut duct with a section of catheter tied in it to prevent its constriction by the purse string suture which holds it in the duodenum will at times, I believe, be safer than direct suture between the end of the duct and the duodenum.

The production of an external biliary fistula and its implantation, while an undesirable operation and one in which many failures and recurrences of stricturing will occur, will occasionally need to be practiced as a last resort.

Careful preliminary isolation of the cystic artery and its clamping and ligation before the cystic duct is cut in cholecystectomy will prevent most

of the cystic or hepatic artery hemorrhages in cholecystectomy, which in turn will prevent duct injury and the production of duct strictures.

Finally, there must be no more distressing situation in surgery than that associated with the production of an hepatic or common duct stricture. There is no result in surgery which is more gratifying than the successful operative relief of this condition and there is no place in surgery where surgical judgment and technical skill can better be exhibited than in the operative management of strictures of the common and hepatic ducts.

DISCUSSION OF THE PAPERS OF DOCTORS HEUER AND LAHEY

DR. ROBERT L. PAYNE (Norfolk, Va.).—I know there are many men here who are far more capable of discussing these papers than I am, but I wish to indulge myself in discussing particularly Doctor Heuer's paper, because of my interest in acute cholecystitis. I do not wish to discuss when to and when not to operate on acute cholecystitis, but to summarize my attitude in saying that, given a case we recognize as acute cholecystitis, if the signs and symptoms are progressive, or if a retrogression in this clinical picture is followed by pain and leukocytosis, I think operation is immediately necessary. In anticipation of this discussion on acute cholecystitis, I hurriedly reviewed 355 gallbladder cases we had operated upon in the last nine years and found that 65 cases were acute cholecystitis, and of those 29 were gangrenous.

I probably feel differently than many of you in that I am strongly opposed to cholecystostomy in acute cholecystitis. I think in that way we have not removed the cause of the trouble and that the further damage to the liver will continue and cause more trouble than operative removal. Gangrene occurred in this group in 8 per cent of the cases, whereas acute cholecystitis was only present in 18 per cent. I do not think any of you would operate on a gangrenous appendix by putting a tube in its lumen and getting out, and I feel that the same applies to acute cholecystitis. We had two deaths in this series but no peritonitis. The only cause of death we could find was acute necrosing hepatitis and I believe that is what occurs in acute cholecystitis when you do not remove the cause.

I was delighted to hear Doctor Lahey speak of planting tubes in the common duct. I have two patients at present with tubes, that have not come away, in the duodenum. One was a very short anastomosis located just where the common duct emerges from the liver. The anastomosis was effected over a tube and in that case, instead of the tube working down as they usually do, it went up into the liver. It can be seen in the roentgenogram. I hesitate to go in and remove it and wish Doctor Lahey would help me out with some suggestions.

DR. FRANK K. BOLAND (Atlanta, Ga.).—Doctor Lahey has described several ingenious and skillfully performed operations for the difficult condition of stricture of the bile ducts. I wish to report a ten year old case of bile duct obstruction caused either by stricture or stone, probably the latter. A few days after removal of the gallbladder for stones, there was a copious flow of bile from the drained wound, soon followed apparently by a discharge of the entire biliary secretion. I thus observed the condition of a patient who was losing all of her bile. Of course the stools were absolutely clay colored. Retention of bile in the system, with jaundice, causes itching, and I learned that absence of bile causes intense, intolerable itching. The

patient's suffering became so severe that she had to be restrained in the bed. She could not eat, vomited incessantly, and became markedly emaciated. The pruritus was especially severe in the mucous membranes, which were vermilion red. I could collect most of the bile through a drainage tube, and attempted to reintroduce it into the patient through a duodenal tube, but she permitted the procedure only once. In those days we were not giving as much saline and glucose as we are today. The patient's condition was so bad that I did not think she would stand any prolonged manipulation to close the biliary fistula, so I adopted the following measure: Under local anesthesia a jejunostomy was performed through the left rectus muscle. I then had a rubber tube leading into the jejunum, and another rubber tube draining bile from the fistula. These two tubes were connected outside the body by a glass tube. I then could observe bile flowing out of the body, and back into the body again. This flow continued for only three days, during which time all the patient's symptoms abated, and she improved rapidly. I then removed the drainage tube, but fed the patient through the jejunostomy tube for ten days, when the tube came away. I cannot tell in what manner this operation helped the patient, except that it preserved her bile, and saved her life. Evidently it removed the obstructing agent, whatever it was. Her weight had dropped to 100 pounds. I saw her a few months ago. She was in good health and weighed 200 pounds.

DR. WALTMAN WALTERS (Rochester, Minn.).—These papers on lesions of the biliary tract are outstanding contributions to our knowledge of this field. Time will not permit commenting on more than one of them and so I will confine my remarks to discussing Doctor Lahey's paper.

He is to be congratulated on his choice of surgical procedures employed in the cases which he has presented. The successful result which has followed employment of this variety of surgical procedure in the treatment of stricture of the common bile duct illustrates the fact that each case is an individual problem in which the surgeon is concerned with restoration of continuity between normal tissues beyond the stricture. In my experience the operation of choledochoduodenostomy or hepaticoduodenostomy has given the best results, providing it is possible to make an accurate anastomosis. Yet experience has shown that good results also have been obtained in the few cases in which local excision of strictures has been carried out with anastomosis of the end of the duct in cases in which the stricture was situated so high on the common duct that insufficient duct remained above the stricture to permit choledochoduodenostomy to be performed, because of the inability to mobilize the duodenum satisfactorily, and in some cases of complete stricture of the extrahepatic bile ducts in which external biliary fistulae have been transplanted into the stomach.

In elaborating further on the results following these and other procedures, I think you would be interested to have a later report of the 29 cases of stricture of the common bile duct which I reported before this Society in 1931.

An additional five years have elapsed since that report. My former first assistant, Dr. K. K. Nygaard, now associated with Semb, of Oslo, Norway, and I have completed a study of these 29 cases as well as an additional 22 cases upon whom I have operated since that time. In his[1] report he said: "In this series of twenty-nine cases of stricture of the common bile duct in which operation was performed by Walters of The Mayo Clinic there were four deaths in the hospital, or an immediate mortality of 13.8 per cent. By correspondence or reexamination at the Clinic I have been able to get in-

formation regarding the subsequent course of all the other patients. At the time this was written sixteen patients were still living and fourteen of them or about 48.3 per cent of the entire group, were in good physical condition (Table I). One of these patients, a woman who at the time of her operation was thirty-two years of age, went home in excellent condition and went through a perfectly normal pregnancy and delivery one year after operation. Nine patients were found to have died subsequent to operation. From letters received, eight of these deaths appeared to have been directly attributable to the original pathologic lesion in the strictured bile duct and to associated damage to the liver. In one case the cause of death was bronchopneumonia. Ten years was the longest period and twenty months the shortest period that any of these nine patients had lived after operation. The average duration of life was five years and three months. The average age of these nine patients at the time of operation was forty-two years. The normal expectancy of life at this age is eighteen to twenty years. Since the expectancy of life for the group of patients under consideration, if operation had not been performed, was limited, life in these nine cases appears to have been prolonged to about 20 to 25 per cent of the normal expectancy. It is interesting to meditate what this percentage of prolongation of life will be when a full account can be given of the entire group of cases in the future.

TABLE I

RESULTS, AS OBTAINED BY A RECENT FOLLOW UP STUDY OF THE 29 CASES IN WHICH OPERATION FOR STRICTURE OF THE BILE DUCTS WAS PERFORMED PRIOR TO 1933 BY DOCTOR WALTERS

Result	Cases	Per Cent
Deaths in hospital	4	13.8
Subsequent deaths*	9	31.0
Living and in good condition	14	48.3

* The average duration of life after operation of these nine patients was five years and three months.

"Since January 1, 1932, Dr. Walters has performed operations in twenty-two additional cases of stricture of the bile ducts, making a total of fifty-one cases. Of these twenty-two patients, three died in the hospital, giving an immediate mortality of 13.6 per cent, which is practically identical with the hospital mortality for the group of patients operated on before 1933. In four of these cases the immediate cause of death was postoperative cholemic hemorrhage. The fifth patient, a woman fifty-two years of age with a complete stricture of the common and hepatic ducts on whom a last stage transplantation of external biliary fistula had been performed, did nicely for one week. On the eighth postoperative day, however, she had a massive hemorrhage necessitating opening and packing of the wound. She never recovered after this and died about two and a half weeks after operation. In the sixth case bleeding from the wound and gastro-intestinal tract was a serious complication during the second postoperative week. The bleeding stopped, however, when the value for blood urea started to increase and edema developed in her extremities. The patient died three and a half weeks after operation from hepatic insufficiency and uremia. The immediate cause of death in the seventh case was hepatic insufficiency.

"It seems obvious, therefore, that the greatest danger in these cases of more or less long-standing stricture of the bile ducts is postoperative cholemic hemorrhage.

"Of the remaining nineteen patients, I have recent information indicating that fifteen, or about 68.2 per cent of the original twenty-two are living and in good condition. Of the remaining four patients, one died at home from abdominal hemorrhage brought about by recurrence of the lesion in the biliary tract and the other three are living but are not in good condition."

REFERENCES

[1] Nygaard, K. K., Shelden, C. H., and Walters, Waltman: Benign Strictures of the Bile Ducts: Results in Fifty-one Operative Cases. Proc. Staff Meet., Mayo Clinic. (In press.)

[2] Walters, Waltman: Resections of the Common and Hepatic Bile Ducts and Ampulla of Vater for Obstructing Lesions: Results in Thirty Cases. Surg., Gynec., & Obstet., **56**, 235–241, February, 1933.

DR. RAYMOND P. SULLIVAN (New York, N. Y.).—Stricture of the common or hepatic bile duct, with or without fistula, is a vexatious and important clinical problem because (a) of its own variation in anatomic difficulties and (b) the consequential pathologic changes incident to its presence, prone to affect liver and blood structures. Doctor Lahey has enumerated the fundamental principles involved in the surgical management of these cases. I desire to call attention, by emphasis, to a possibility of reformation of common duct stricture resulting from secondary fibrosis at the site of repair of an original stricture. To illustrate, the appended case is cited.

Case Report.—A. B., a female, age 33, married, was admitted to St. Vincent's Hospital, New York, July 2, 1931, complaining of recurrent attacks diagnosed, after study, as due to cholecystitis and probable calculi. Cholecystectomy with drainage and appendicectomy were performed. The surgeon noted that: "Because of the apparent disease of gallbladder, thickened and surrounded by adhesions, and the pouchy condition of the portion near the cystic duct—gallbladder was removed and liver sulcus drained." Postoperative Course: Returned from operation in good condition. Color good. Blood pressure 124/74. Pulse 110. On second postoperative day dressings were soaked with bile, necessitating frequent changes daily. Drain and stitches were all out by the tenth day. By the twenty-first postoperative day, discharge of bile was copious. Stools were clay colored. At this time I saw the patient for the first time, and concurred in the opinion that the biliary fistula was due to common duct injury associated with stricture or impacted calculus. Operation was advised but not accepted until August 26, the forty-eighth postoperative day. Operation, August 28, 1931. The opening from which bile was exuding was located in the portal fissure close to the junction of the left and right hepatic ducts. Slight traction on the fibrous common duct stopped the flow of bile and revealed the opening to be in the right hepatic duct.

The procedure was to excise the stenosed portion of the common duct and to dilate the fistulous opening near the fusion of the hepatic ducts sufficiently to include both hepatic ducts. Then a V shaped flap from the duodenal wall was made in a transverse direction. The apex was fixed to the ductal opening by interrupted sutures on the posterior wall. A T tube was inserted. The margins of the duodenal walls and duct were closed over arms of T tube. The approximation was satisfactory and without tension. Additional drains were placed alongside stem of T tube. Wound was closed. Postoperative course was stormy for three days. Bile passed into the intestinal tract, and was noted in the stool on fifth day. Auxiliary drains and stitches were all out by the twelfth day and progress was satisfactory. The T tube stem was clamped off by rubber band intermittently. General improvement continued. However, on October 10, the intern reported absence of T tube. Roentgenologic examination

revealed the tube in the descending colon, from which it was passed October 14 without distress. The abdominal wound healed rapidly and the patient was discharged October 24, 1931, free of symptoms. She was observed at follow up clinic at regular intervals until January 30, 1932. When seen March 31, 1932, she reported having had an attack of "grippe" five weeks previously—following which she noted beginning jaundice and clay colored stools. She refused operation at this time and was treated medically. However, she was readmitted, May, 1932, and reoperated upon, June, 1932. A definite stricture was found at the site of the previous anastomosis between duct and duodenum. The hepatic duct was now longer than previously. The procedure was to divide the stricture on its anterior aspect leaving the posterior attachment intact. Bile gushed out with considerable force. It was possible to keep the operative field clear by continuous suction. A T tube was inserted into the right hepatic duct and duodenum and reconstruction made over the transverse arms of the tube. Secondary drains were placed to either side of the site of anastomosis and the wound was closed.

The postoperative course was most satisfactory. The jaundice cleared rapidly and the wound healed quickly. All auxiliary drains and stitches were removed on twelfth day. The patient was discharged July 17, 1932, with instructions as to how to manage the T tube stem. On January 23, 1933, or six months postoperatively, the patient had gained 40 pounds and was free from all symptoms of distress. Stools were normal in color and consistency. She now requested removal of T tube, which was done January 25, 1933. She was discharged February 18, 1933, with wound completely healed and no complaints or distress. She was seen at stated intervals with happy reports. When last seen, October 20, 1936, four years after second operation, her condition was satisfactory.

This case illustrates the necessity for accurate anatomic recognition when performing cholecystectomy. Secondly, that injury to the common duct will usually not be recognized at time of operation. Thirdly, that successful reconstruction of the common duct may be followed by secondary stricture.

DR. W. D. HAGGARD (Nashville, Tenn.).—I will speak briefly of a simple and satisfactory method of caring for the cases of gangrene of the gallbladder which are so severe and difficult. They are so water-logged and friable that one has great difficulty in doing a cholecystectomy. These cases give the greatest danger of injury of the ducts because it is so difficult to expose them. Inadequate exposure makes accidents happen. I may say that this method is not original with me but was first suggested by Dr. Denegre Martin and also independently by Doctor Gatch. The object is not to attempt to strip the gallbladder from its bed in the liver, which entails so much bleeding, but instead to remove all of the gangrenous gallbladder, leaving only the healthy posterior wall of the gallbladder in the fossa of the liver. After that, one can destroy the remaining mucosa by one of two methods, either by the use of carbolic acid on a gauze sponge or by means of a cautery. Then one introduces a catheter into the cystic duct for drainage and sutures the healthy cut edge of the wall of the gallbladder over it. This method simplifies and makes possible the quick and safe removal of a gangrenous gallbladder. I wish to commend it as being an excellent way to accomplish what is otherwise a difficult cholecystectomy.

THE SURGICAL TREATMENT OF CHRONIC BILIARY TYPHOID CARRIERS

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IT IS generally agreed that the carrier problem is the most important one remaining in the eradication of residual *Bacillus typhosus* infection. Unfortunately nothing has been accomplished toward preventing a typhoid fever patient from becoming a typhoid carrier, nor, indeed, has much attention been paid to this problem in preventive medicine. Public health practice has concerned itself, instead, with the discovery, control and cure of persons in whom the carrier state has been established.

Since the establishment of typhoid fever stations in Germany at the beginning of the century, many therapeutic agents have been used in the treatment of carriers. Stertenbrink[1] reviewed the literature on the subject and called attention to the many unsuccessful attempts that had been made up until 1928. Furthermore, since then there has been no evidence of an effective curative agent except that of Gulbrandsen,[2] who reported four cures in 12 chronic carriers treated with deep roentgen therapy over the gallbladder. However, Forsbeck and Lampe[3] were unable to effect any cures in a series of cases treated with deep roentgen therapy, although negative specimens became more frequent during treatment in some instances.

In 1888, Anton and Fütterer[4] isolated the *B. typhosus* from the gallbladders of fatal typhoid fever cases. Later, in the German carrier campaign it was discovered that the organism could usually be found in the gallbladders of chronic carriers. Indeed, as was pointed out by Mallory and Lawson,[5] in 1931, the persistent infection of the gallbladder in typhoid carriers usually produces a chronic inflammation of that organ which is characteristic though not pathognomonic. It has long been known that cholelithiasis is usually present in chronic typhoid carriers.

In 1908, Grimme[6] reported a cholecystectomy performed for the purpose of curing a carrier. Since that time, this method has become an accepted, though not very common, therapeutic procedure for the cure of gallbladder carriers. In the reference of Senftner and Coughlin[7] to the work of Bergglas[8] and to that of Hage and Brinkmann,[9] there is an overcount of operations performed. Of the 74 cases of Bergglas, only two were his own, 66 of the others being those already summarized by Hage and Brinkmann.

The benefits of cure by cholecystectomy accruing to the carrier, his family and the community may be summarized as follows:

(1) The occasional case that occurs even after a person knows himself to be a typhoid carrier is prevented.

(2) The former food-handling carrier is able to resume his occupation.

(3) The mental depression frequently observed in carriers is relieved.

(4) Potential or actual gallbladder disease is eradicated.

(5) The health department is released from the expense of controlling a potential source of infection.

Eligibility Criteria for Operation and for Release of Chronic Biliary Typhoid Carriers

Analysis of Previous Reports.—In the early work subsequent to Grimme's report, neither preoperative nor postoperative bile specimens were obtained, and release following cholecystectomy was based entirely upon the examination of feces. Many carriers were included in these reports who had submitted to operation because of their clinical symptoms but who were not separately classified either as to cure rates or fatality rates. Furthermore, the literature, especially foreign literature, includes many carriers of the paratyphoid group which are not separately analyzed as to fatality rates and cure rates. Finally, many of the reports include, without separate consideration, convalescent carriers who may be positive for only a few months after illness. Such a carrier has an excellent chance of clearing up spontaneously.

We have omitted the early German literature in preparing Table I because, as Bigelow and Anderson[10] have pointed out, the eligibility criteria for operation and release in the earlier work were not up to the present standards. We have excluded paratyphoid carriers, typhoid carriers who have not had at least one release bile specimen in addition to release feces specimens, convalescent carriers, and persons treated otherwise than by cholecystectomy. We have included, however, cases irrespective of whether or not a preoperative bile was obtained. This analysis reduces the number of cases which can be used for a study of the effectiveness of cholecystectomy to a rather meager number.

Omitting our own series, it may be noted that about 62 per cent of all cases were returned to their communities alive and noninfectious. But if only those who were operated upon because they were carriers are considered, it may be noted that about 74 per cent were cured; and omitting the three deaths occurring in the Senftner and Coughlin series, 79.5 per cent of the cases were cured.

Determination of Gallbladder Infection.—While carriers in whom the only nidus of infection is in the intestinal wall are rare, there is little evidence as to the relative occurrence of this type of carrier. Garbat[11] found one intestinal carrier in 146 convalescent carriers. This, however, cannot be accepted as evidence of the occurrence of intestinal carriers among chronic

carriers.* It was not until after the development of practical duodenal drainage by Einhorn[12] that preoperative bile specimens were commonly obtained. During the past ten years, preoperative diagnostic bile specimens have been usually considered prerequisite to operation in chronic carriers essentially free from gallbladder symptoms. The procurement of a negative specimen (indicating a negative biliary tract) with positive feces would contraindicate cholecystectomy.

It has been shown, by Forsbeck and Hollon,[13] that the reliability of a negative report on a bile specimen depends largely upon the hydrogen ion concentration of the specimen. A mixture of gastric juice and bile is usually submitted to the laboratory by the uninitiated. Such a specimen is usually highly acid. In it the typhoid organisms may be killed in less than 30 seconds after mixture has occurred. Consequently, when a negative specimen of "bile" is obtained preoperatively, it must be known definitely that the specimen is actually bile, before the case may be classified as an intestinal carrier.

In spite of the rarity of intestinal carriers it is not justifiable to subject a carrier to cholecystectomy without definitely knowing that he is a chronic bile carrier, unless the operation is indicated purely from the clinical viewpoint. Recently an illustrative case came to our attention. This person, after two positive feces, had a long series of negative feces. Without a preoperative bile drainage, she submitted to cholecystectomy on the assumption that she was a carrier. No bile was taken for culture at the time of operation. The case was probably a contact feces carrier who cleared up spontaneously before the date of operation.

On the other hand, intestinal carriers are so rare that for the purpose of analysis we may consider that feces carriers are gallbladder carriers, unless proved to be intestinal carriers by the procurement of a negative diagnostic bile specimen. This can, at most, cause but a trifling error in an analysis.

Surgical Risk.—The literature is not clear concerning the mortality rate from cholecystectomy in carriers since it is not always stated whether a carrier submitted to operation mainly to cure his carrier condition, or because of the urgency of his clinical symptoms. To deprecate surgical intervention for the cure of the carrier state because of deaths occurring among carriers operated upon because they were seriously ill with gallbladder disease is obviously illogical.

In so far as surgical risk is concerned each case must be judged without regard to his carrier state, although the Michigan Department of Health as a matter of public policy does not urge cholecystectomy for any person over 50 without symptoms of gallbladder disease. On the other hand, it is considered good surgical practice to urge any good risk with gallbladder pathology to submit to cholecystectomy simply for his own welfare. Cholecystitis

* Several large states and cities have agreed on arbitrarily declaring those who have harbored the *B. typhosus* for one year or more from date of onset with typhoid fever to be chronic carriers, those for less than one year convalescent carriers.

TABLE I

RESULTS OF CHOLECYSTECTOMIES PERFORMED UPON CHRONIC BILIARY TYPHOID CARRIERS

*Summarized from Data of Various Authors**

Author	Year	Percentage of Cures								Mortality Percentage							
		Reason for Cholecystectomy: Gallbladder Symptoms or a Carrier				Reason for Cholecystectomy: Because a Carrier				Reason for Cholecystectomy: Gallbladder Symptoms or a Carrier				Reason for Cholecystectomy: Because a Carrier			
		Recovered or Died		Recovered Cases Only		Recovered or Died		Recovered Cases Only		All Ages		Under 50		All Ages		Under 50	
		No. Operations	Per Cent Cured	No. Operations	Per Cent Cured	No. Operations	Per Cent Cured	No. Operations	Per Cent Cured	No. Operations	Mortality Per Cent	No. Operations	Mortality Per Cent	No. Operations	Mortality Per Cent	No. Operations	Mortality Per Cent
Haaland and Haaland[15]	1927	1	0.0%	1	0.0%	1	0.0%	1	0.0%	1	0.0%	1	0.0%	1	0.0%	1	0.0%
Bergglas[8]	1929	2	100.0%	2	100.0%	2	100.0%	2	100.0%	2	0.0%	2	0.0%	2	0.0%	2	0.0%
Whipple[16]	1929	2	100.0%	2	100.0%	2	100.0%	2	100.0%	2	0.0%	1	0.0%	2	0.0%	1	0.0%
Vogelsang and Haaland[14]	1931	2	0.0%	2	0.0%	2	0.0%	2	0.0%	2	0.0%	0	—	2	0.0%	0	—
Senftner and Coughlin[7]	1933	36	50.0%	28	64.3%	24	66.7%	21	76.2%	61	13.1%	32	0.0%	42	7.1%	26	0.0%
Bigelow and Anderson[10]	1933	8	100.0%	8	100.0%	7	100.0%	7	100.0%	8	0.0%	4	0.0%	7	0.0%	4	0.0%
Hanssen[17]	1935	4	100.0%	4	100.0%	4	100.0%	4	100.0%	4	0.0%	?	—	4	0.0%	?	—
Subtotals	1927–1935	55	61.8%	47	72.3%	42	73.7%	39	79.5%	80	10.0%	40	0.0%	60	5.0%	34	0.0%
Coller and Forsbeck	1937	18	88.9%	18	88.9%	16	87.5%	16	87.5%	18	0.0%	15	0.0%	16	0.0%	14	0.0%
Totals	1927–1937	73	68.5%	65	76.9%	58	77.6%	55	81.8%	98	8.2%	55	0.0%	76	3.9%	48	0.0%

* In abstracting this material, paratyphoid carriers, convalescent carriers, and persons released without at least one negative bile specimen following cholecystectomy have been omitted.

is so nearly universal in chronic carriers and cholelithiasis so common, that for practical purposes it is logical to assume preoperatively the presence of gallbladder pathology. As may be noted in Table I, no deaths have been reported in any series except that of Senftner and Coughlin. In their series the fatality rate was 14.7 per cent for all cases. Of those submitting to cholecystectomy primarily for cure of the carrier state, only three or 7.1 per cent died as a result of the operation. In a personal communication Doctor Senftner has kindly supplied us with the following data concerning these three cases:

Case 1.—P. G., male, age 51. Operated upon, September 20, 1929. Died, September 23, 1929, of acute peritonitis.

Case 2.—M. G. female, age 57. Operated upon, August 12, 1922. Died, August 12, 1922, of surgical shock and acute dilatation of heart.

Case 3.—H. D., female, age 60. Operated upon, June 21, 1932. Died, July 3, 1932, of enterocolitis, toxemia, cholecystitis, cholelithiasis. Convalescing uneventfully until five days after operation.

It may be noted that all these persons were over 50 years of age. Cases 1 and 2 were inmates of state institutions for the insane, persons notoriously of low resistance.

Percentage of Cure.—In determining the percentage of cure, it is proper to include persons operated upon for clinical symptoms, provided this group is analyzed separately. While not yet proved, it would seem almost a truism, that the severity of clinical symptoms would be correlated with the extent of biliary tract involvement and therefore with the chance of cure. Vogelsang and Haaland[14] have suggested the probability that the rate of cure depends upon the number of years that the carrier has had gallbladder infection. Two of their carriers, of respectively 17 and 45 years' standing, were not cured by cholecystectomy. Sufficient accumulation of data may enable us to predict fairly closely the probability of cure based upon the duration of the carrier state.

Present Eligibility Criteria for Operation and Release of Chronic Biliary Typhoid Carriers in Michigan

(A) Eligibility criteria for carriers to be operated upon primarily for cure of the carrier state:

(1) The person must be a chronic carrier.

(2) The person must be a bile carrier.

(3) The person must be a good surgical risk.

(B) Eligibility criteria for release after cholecystectomy:

(1) Twelve consecutive feces specimens obtained at monthly intervals following operation and examined in a laboratory approved by the Michigan Department of Health shall be negative.

(2) Two consecutive bile specimens obtained during the year after operation and considered satisfactory for examination by the Michigan Depart-

ment of Health shall be examined in an approved laboratory and found to be negative.*

Results of Cholecystectomy in Michigan Series.—Our series of 18 cases has been summarized in Table II. Eleven of these cases were operated upon at the University Hospital.† All cases were chronic carriers. Twelve, or 67 per cent, had slight to marked symptoms of gallbladder disease. In two of these (Cases 5 and 16), operation was performed primarily for the relief of clinical symptoms. No deaths occurred in our series.

Of 11 preoperative bile specimens obtained ten were positive, while of 15 bile specimens obtained at operation 14 were positive. The negative preoperative bile specimen was obtained by an observer unfamiliar with the necessity of securing bile and not a gastric juice-bile mixture. The negative bile specimen obtained at operation was examined in a laboratory which had been doing but little typhoid work. In Case 1, only, it was not definitely proved by a positive bile that the person was a bile carrier. However, the patient was cured by cholecystectomy.

Of the 18 cases, three were males and 15 females. The average age at operation for the males was 35, for females 40.3. The total age incidence was 39.4. Two gave no history of having had typhoid fever. Of those giving a positive history, the duration of infection had varied from one to 38 years, with an average of 15.2 years.

The percentage of cures was 88.9 per cent. Two carriers were still feces-positive, one 14 and the other 15 months after operation. The former (Case 11) had had typhoid fever 28 years before operation, while the latter (Case 13) gave no history of having had the disease. Case 13 is still a biliary tract carrier as indicated by a positive bile obtained 15 months after operation. No postoperative bile has been obtained from Case 11. Case 6, a graduate nurse employed in a hospital, became feces-negative promptly after operation. The first postoperative bile was negative, but this was followed 18 months after operation by a positive finding. Twenty-six months after operation a third bile specimen was negative. During the two years since operation 16 negative feces specimens and no positives have been obtained. The first bile specimen obtained in the hospital where she works was not considered satisfactory by the Michigan Department of Health. We believe that this patient may have been reinfected or that she maintained a slight nidus of infection which was missed in the first bile specimen.

As can be seen in Table II, in most instances the feces became negative within a few days to three weeks after operation. However, the feces may

* If further evidence should confirm previously accumulated data,[13] it is probable that the Michigan Department of Health will change the number of negative release biles required from two to one. Already, because of unusual circumstances, two carriers have been released with 12 negative feces and one negative bile.

† We are indebted to Drs. M. J. Darling, W. R. Clinton, and F. B. Wight, of Detroit, F. J. O'Donnel of Alpena, and C. B. Wood of Clare, each of whom has had one case under his care at other hospitals.

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TABLE II

RESULTS OF CHOLECYSTECTOMIES PERFORMED UPON CHRONIC BILIARY TYPHOID CARRIERS IN MICHIGAN

Case No.	Sex	Age at Oper.	Years Since T. F.	Symptoms Suggesting Gallbladder Disease	Gallbladder Pathology			Diagnostic Biles		Bacteriology Release Specimens		
					Chronic Cholecystitis	Stones	Other	Preoper.	At Oper.	Biles	Feces: Days Pos. after Oper.	Feces: No. of Negs. after Last Pos.
1 V. R.	F.	30	9	Bilious attacks every 3–6 mos. from T. F. to time of oper. None in 10 mos. following	Yes	No	—	None	Neg.	2 negs.	0	13
2 V. P.	F.	26	9	None	Yes	Yes	—	Pos.	Pos.	2 negs.	308	16
3 F. L.	F.	36	2	None	Yes	Yes	—	None	Pos.	2 negs.	64	13
4 A. K.	F.	54	8	Occasional sharp pain and tenderness, U. R. Q. Dull aching pain, U. R. Q. since T. F.	Yes	No	—	Pos.	None	2 negs.	9	20
5 L. B.	F.	46	?	Bilious attacks frequently. Recurrent attacks U. R. Q. Pain for past 8 yrs.	Yes	Yes	Greatly enlarged	None	Pos.	2 negs.	9	15
6 L. H.	F.	36	13	None	Yes	No	—	None	Pos.	1 N. 1 P.	0	16
7 E. C.	F.	42	2	None	Yes	Yes	—	Pos.	Pos.	1 N. 2 negs.	0	16
8 D. S.	F.	44	36	Severe G. B. attacks 1929–1933. Many attacks of U. R. Q. pain. Occ. clay colored B. M.	Yes	Yes	Yes*	None	Pos.	1 neg.	0	3
9 A. W.	M.	51	16	None	Yes	Yes	—	Pos.	Pos.	2 negs.	29	23
10 T. K.	M.	39	18	None	No	Yes	—	Pos.	None	1 neg.	0	2
11 E. M.	F.	39	28	Attack of jaundice 18 yrs. ago	Yes	Yes	Yes*	Pos.	Pos.	None	433	0
12 D. W.	F.	27	2	Severe acute G. B. attacks month before oper.	Yes	No	—	Neg.	Pos.	2 negs.	0	19
13 G. S.	F.	44	?	Tenderness, U. R. Q.	Yes	Yes	2 times normal size	None	Pos.	1 pos.	460	0
14 C. A.	M.	15	1	G. B. symptoms during illness with typhoid fever	Yes	Yes	—	Pos.	None	1 neg.	0	14
15 M. J.	F.	55	38	Bilious attacks 25 years. Tenderness U. R. Q.	Yes	Yes	—	Pos.	Pos.	2 negs.	0	26
16 G. E.	F.	48	22	Pain and tenderness U. R. Q. progressively worse 13 years previous to oper.	Yes	No	—	None	Pos.	2 negs.	0	12
17 I. G.	F.	33	2	No G. B. symptoms past 10 yrs. About 1 attack per year previously	Yes	Yes	—	Pos.	Pos.	1 neg.	0	10
18 T. S.	F.	45	37	Treated for cholelithiasis 6 mos. before oper. Frequent U. L. Q. pain	Yes	Yes	Hepatitis	Pos.	Pos.	2 negs.	16	14

* Active chronic, purulent cholecystitis.

remain positive for a much longer time and eventually become negative, as is shown in Case 2. For two months this patient continued to submit stool specimens in spite of the fact that the specimens remained positive. Because of our inexperience, we were convinced in our own minds that this was to be a failure; however, after 308 days the feces became negative and have remained so.

Twelve patients had symptoms characteristic of cholecystitis or cholelithiasis while the remaining six patients had no symptoms even suggestive of disease of the biliary tract. Gallstones were present in 13 patients and absent in the remainder. There was a nearly uniform finding in the gallbladder on pathologic examination of chronic cholecystitis with marked lymphoid hyperplasia. In two instances, active, chronic, purulent cholecystitis was present and in two cases the gallbladder was greatly enlarged but with the common finding of chronic cholecystitis.

It is worthy of emphasis that a third of the patients had no symptoms referable to the gallbladder, nearly one-third had no stones and the changes in the gallbladder were nearly all minimal in character. In every case, however, there did exist definite changes in the gallbladder characteristic of long standing low grade infection, and aside from the cure of the carrier state, so desirable from the public health standpoint, we feel that cholecystectomy will usually improve the health of the patient to such an extent as to make it very much worth while also from the viewpoint of the individual.

SUMMARY

(1) In our series, cholecystectomy was performed on 18 chronic biliary typhoid carriers with a percentage of cure of 88.9.

(2) No deaths occurred in the series.

(3) Eligibility for cholecystectomy was based on the carrier being a gallbladder carrier and a good surgical risk.

(4) Cure was considered effected when 12 consecutive negative feces specimens and at least one (usually two) negative bile specimens were obtained.

(5) Most cases become feces-negative in from a few days to two or three weeks after operation. Hope for eventual cure may be maintained for many months, however.

(6) Mortality and cure percentages are comparable only if:

a. Typhoid and paratyphoid fever carriers are considered separately.

b. Carriers operated upon primarily for cure of the carrier state and primarily for clinical symptoms are considered separately.

c. The time elapsed since typhoid fever is taken into consideration.

d. The criteria for release are identical or at least similar.

(7) Cholecystectomy may logically be recommended as a matter of personal precaution to a chronic carrier without clinical symptoms who is a good risk.

(8) Each cure removed one more of a limited number of infection foci and thus contributes to the ultimate eradication of typhoid fever.

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ACUTE PERFORATED APPENDICITIS WITH PERITONITIS

REPORT OF TWO HUNDRED FIFTY-TWO CONSECUTIVE CASES *

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IN THIS paper, 252 consecutive cases of acute perforated appendicitis treated by members of the surgical department of the Henry Ford Hospital during the years 1915 to 1933 are studied, with special consideration of factors contributing to, and responsible for, the mortality rate. Our experience has been that in 22.1 per cent of patients admitted with acute appendicitis, perforation has occurred.

TABLE I

Type of Peritoneal Involvement	Group	No. of Cases	Percentage of Total Cases	Percentage of Mortality Rate
Local peritonitis	A	55	21.83%	0%
Abscess	B	117	46.43%	4.2%
General peritonitis	C	65	25.79%	21.5%
General peritonitis plus abscess	D	15	5.95%	46.6%
Totals		252	100.00%	10.4%

Of the 252 cases, 21.8 per cent of early perforations were complicated by local peritonitis only, with a mortality rate of zero; 46.4 per cent by circumscribed abscess formation with an operative mortality of 4.2 per cent;

TABLE II

ACUTE APPENDICITIS, 1933–1934

Type	No. of Cases	No. of Deaths	Mortality Rate
Acute diffuse	87	0	0%
Acute diffuse (drained)	25	0	0%
Acute gangrenous (drained)	34	0	0%
Acute perforated with local peritonitis (drained)	26	0	0%
Acute perforated with abscess	22	2	9%
Acute perforated with diffuse peritonitis	11	1	9%
Totals	205	3	1.5%

25.7 per cent by acute spreading or generalized peritonitis with an operative mortality of 21.5 per cent; and 5.9 per cent by both acute generalized

* Dr. Clyde Allen has operated upon many of these patients and helped with the preparation of the paper.

peritonitis and abscess formation with a mortality rate of 46.6 per cent. The operative mortality of the entire series was 10.4 per cent. Two cases of general peritonitis in extremis were not operated upon; therefore the total mortality was 11.1 per cent. The mortality rate of all cases (205) of acute appendicitis, both ruptured and unruptured, during 1933 and 1934 at this clinic was 1.5 per cent.

Considering the operative mortality rate in the ruptured cases for the five year period from 1915 to 1930, we find that there was a steady decrease from 22.2 per cent to 11.3 per cent. In the period between 1930 and 1934, it had further decreased to 7.7 per cent.

TABLE III

SHOWING DECREASE OF OPERATIVE MORTALITY RATE DURING 19 YEAR PERIOD OF RUPTURED APPENDICITIS, 1915–1933*

Period in Years	No. of Cases	No. of Deaths	Operative Mortality Rate
1915–1919	9	2	22.2%
1920–1924	31	4	12.9%
1925–1929	96	11	11.4%
1930–1934	116	9	7.7%
Totals*	252	26	10.4%

* A recent survey of 85 cases of acute perforated appendicitis from January, 1934, to June, 1936, shows an operative mortality rate of 4.7 per cent.

PREDISPOSING ETIOLOGIC FACTORS.—*Sex.*—One hundred seventy-one cases, or 68.4 per cent of the entire series, were males and 79 or 31.6 per cent were females, a ratio of slightly more than two to one.

Age.—Acute appendicitis is usually a disease of young individuals. Between the ages of ten and 30 years is by far the most common period for the initial occurrence of appendicitis. When the four groups of peritoneal involvement are considered separately, however, quite a different picture is obtained. The average age of the local peritonitis group was 27.7 years and the average age of the generalized peritonitis group was 27.5 years. In the group complicated by appendiceal abscess, the average age was 40.3 years and in the group with abscess and generalized peritonitis the average age is 38 years. Thus there is found to be a consistent difference between the average age in the groups with abscess formation on the one hand and in the groups with local and generalized peritonitis on the other hand. The youngest case in the entire series, one of the general peritonitis, was 20 months of age, while the oldest was 81 years of age, having local peritonitis. Both cases recovered after operation.

Race.—Caucasians comprise the great majority on this series. There were only 29 Negroes, making 11.5 per cent of the total number. Only

TABLE IV

AGE DISTRIBUTION OF CASES PRESENTED

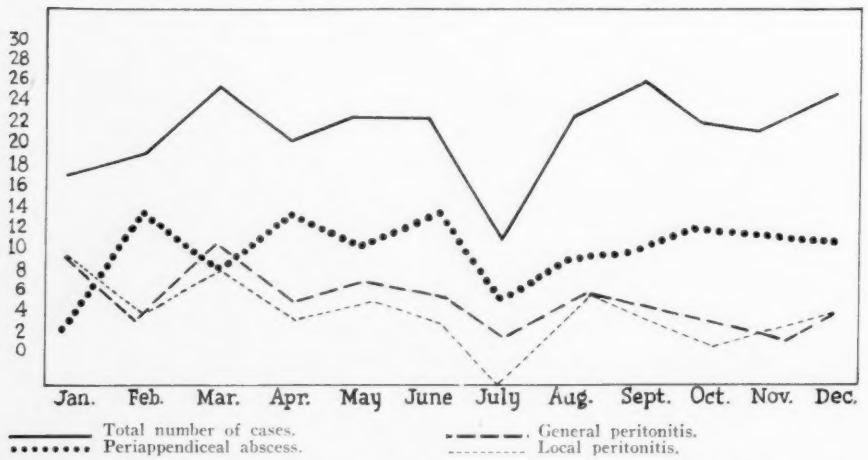
Age in Years	General Peritonitis		Abscess		General Peritonitis and Abscess		Local Peritonitis	
	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent
1–10.....	7	39.0	12	24.0	1	13.3	5	30.9
11–20.....	18		17		1		12	
21–30.....	14	49.0	22	63.0	2	66.6	18	61.9
31–40.....	8		27		6		15	
41–50.....	10		24		2		1	
51–60.....	7	12.0	8	12.0	2	20.0	1	7.2
61–70.....	1		6		1		0	
71–80.....	0		1		1		0	
81–90.....	0		0		0		1	
Totals...	65	100	117	100	15	100	55	100

one member of the Mongolian race is found in the entire series. Twenty-seven nationalities are represented; namely, American, Polish, Scotch, Chinese, Canadian, French, Swedish, Armenian, Jugo-Slav, Italian, Roumanian, German, Lithuanian, English, Austrian, Arabian, Russian, Irish, East Indian, Hindu, Greek, Finnish, Dutch, Danish, Mexican, Maltese and Syrian.

Season.—The greatest number of cases occurred in the months of March, August, September and December, although the cases were fairly well dis-

TABLE V

SHOWING SEASONAL INCIDENCE CURVES OF THE THREE GROUPS OF PERITONEAL INVOLVEMENT



tributed throughout the entire year. There was a uniform and definite remission in the number of cases of general peritonitis, local peritonitis, and abscess in July.

EXCITING FACTORS.—*Foreign Bodies.*—The presence of fecaliths was noted in most of the cases with appendiceal abscesses and this undoubtedly was responsible for the development of the well named obstructive appendicitis. One case at operation presented a straight pin surrounded by hard calcified material lying within the appendiceal lumen having ulcerated through the wall of the appendix into the peritoneal cavity, causing an abscess. In other instances, a hard irregular fecalith had perforated the coats of the appendix and, although the main body still remained within the lumen, small projections from the fecalith presented through one or more small perforations.

TABLE VI

SHOWING THE ORGANISMS CULTURED FROM PERITONEAL PUS AT OPERATION OR AUTOPSY IN 198 CASES OF PERFORATED APPENDICITIS. NO BACTERIOLOGIC REPORT WAS AVAILABLE IN 54 CASES

Organism	Local Peritonitis No. of Cases	General Peritonitis No. of Cases	Abscess No. of Cases	General Peritonitis and Abscess No. of Cases	Total No. of Cases
B. coli	28	41	78	7	154 or 77.7%
Short-chained gram-positive nonhemolytic streptococcus often showing diplo-forms	4	8	29	2	43 or 21.7%
Large gram-positive bacillus	1	3	4	0	8 or 4.0%
B. pyocyaneus	0	2	2	0	4 or 2.0%
Streptococcus viridans	1	0	3	0	4 or 2.0%
Hemolytic Streptococcus	0	0	2	0	2 or 1.0%
B. paratyphosus	0	1	0	0	1 or 0.6%
Diphtheroid	0	1	0	0	1 or 0.6%
Friedländer's bacillus	1	0	0	0	1 or 0.6%
M. catarrhalis	0	1	0	0	1 or 0.6%
Unidentified gram-negative bacillus	0	0	1	0	1 or 0.6%
Negative cultures	8	5	11	2	26 or 13.1%

Hematogenous Infection.—Appendiceal infection is sometimes of hematogenous origin. The association of appendicitis with influenza is well known, having been proved by epidemics, notably that of 1918. Appendicitis immediately following acute upper respiratory infections certainly occurs. Acute attacks occasionally occur immediately after removal of badly infected tonsils. In these hematogenous infections, the invading organism is usually the Streptococcus and less often the Staphylococcus. Rosenow has shown selective affinity in some strains of Streptococci for the appendix when inoculated into rabbits. In this particular series 9.1 per cent of our total cases were immediately preceded by acute upper respiratory infections. The

mortality rate of this group was 12 per cent, two of the cases being fulminating. The onset of acute appendicitis four and five days following tonsillectomy occurred in two cases. One of these cases was fatal.

Focal Infection.—Evidence of focal infection elsewhere in the body was found in 92.3 per cent of the total number of cases. Thirty-six per cent of the cases had dental caries; 34.2 per cent had chronic tonsillitis; 20 per cent had gross pyorrhea; and 9.1 per cent had acute upper respiratory infections.

Trauma.—Many cases of acute appendicitis have been attributed to trauma. It is very doubtful whether the appendix can be injured by any trauma to the abdominal wall. One man in this series received an injury to his right side and abdomen. Following the injury, cramping abdominal pain began in umbilical region with associated nausea but no vomiting. He was unable to sleep and took castor oil. Five days later, he was admitted to the hospital and an acute gangrenous appendicitis with perforation and abscess formation was found at operation. This patient had had no history of attacks of appendicitis previously.

Catharsis.—Twenty-nine point seven per cent of the cases that perforated were given a cathartic prior to hospitalization either by some member of the family or by their physician. Bowers had shown that of those patients with acute appendicitis who took no cathartic, only one in 80 died; of those who took one cathartic, one in 13 died; while of those who took two or more cathartics, one in seven died. In this series of those who took no cathartics, one in 15 died; of those patients who took one cathartic, one in 10.5 died; of those who took two or more cathartics, one in 5.5 died.

Diabetes mellitus was present in seven cases and three of these patients died.

Opiates.—Morphine had been given before hospitalization of the patient in only five cases, or 1.9 per cent, by the family physician. In each instance, a delay in hospitalization and operation occurred.

Bacteriology.—Appendicitis is essentially an inflammation of the appendix due to a bacterial infection regardless of its mode or origin. The appendiceal wall becomes invaded either by organisms present in the appendiceal lumen through some traumatized area in the mucosa, or by organisms carried to it by the blood stream. Of the infecting organisms, investigators have found *B.coli* in pure or mixed culture the most constant. It is to be remembered that *B.coli* is not the only agent causing a putrid odor in peritonitis, the anaerobic organisms, especially anaerobic Streptococcus putrificus, being at least partly responsible in many instances. It has been pointed out by Schottmuller, Frankel and others that some of these anaerobes tend to cause thrombophlebitis in any portion of the body they invade. Witness the presence of the Streptococcus putrificus in thrombophlebitis of the internal jugular vein complicating peritonsillar abscess and less often in pyelophlebitis

complicating appendicitis. In appendicitis pus, Brütt found anaerobic Streptococci and *B.coli* in 45 cases, *B.coli* in pure culture in 36, *B.coli* and other aerobes in eight in a series of 107 cases. In the same series, purulent putrid pus in 15 instances yielded no organisms on culture.

In our series, *B.coli* was found in either pure or mixed cultures in 77.7 per cent of the cases. Nonhemolytic, gram-positive, short-chained cocci (aerobic), often with many of the organisms appearing in diplo-forms, were found in 21.7 per cent of the cases.

Pathology.—The pathology reports show acute gangrenous appendicitis to be the most frequent in this series as well as the most serious. In almost 32 per cent of the cases, the appendicitis was described by Dr. Frank Hartman as "acute gangrenous." One-half of these fatal cases (46.4 per cent) had gangrenous appendices. The seriousness of the acute gangrenous appendicitis is evident in the fact that it was present in 46.0 per cent of those cases with generalized peritonitis, and in 68.7 per cent of the fatal cases of general peritonitis.

TABLE VII

SHOWING PATHOLOGIC TYPES OF APPENDICITIS MORTALITY IN 224 CASES. NO PATHOLOGIC REPORT WAS AVAILABLE IN 28 CASES, AMONG WHICH THERE OCCURRED SIX DEATHS. IN 19 OF THESE CASES THE APPENDIX HAD NOT BEEN REMOVED AT OPERATION

Pathologic Report	Local Peritonitis		Generalized Peritonitis		Appendiceal Abscess		Abscess and General Peritonitis	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
Acute gangrenous	15 (28.8%)	0	29 (68.7%)	11 (68.7%)	27 (27.8%)	2 (40.0%)	0 0	0 0
Acute diffuse	15 (28.8%)	0	12 (19.0%)	1 (6.2%)	15 (15.4%)	0	0	0
("Just") Acute appendicitis	14 (26.9%)	0	10 (16.0%)	1 (6.2%)	38 (37.0%)	1 (20.0%)	8 (11.5%)	3 (42.8%)
Acute ulcerative	1 (1.9%)	0	5 (8.0%)	0	4 (4.0%)	0	0	0
Acute exacerbation of chronic	5 (8.6%)	0	5 (8.0%)	1 (6.2%)	11 (11.0%)	0	3 (23.0%)	1 (14.3%)
Acute purulent	0	0	2 (3.0%)	0	0	0	0	0
Subacute	1 (1.9%)	0	0	0	3 (3.0%)	0	1 (7.7%)	1 (14.3%)
Chronic necrotic (caused by a straight pin)	0	0	0	0	1 (1.0%)	0	0	0
Acute hemorrhagic	1 (1.9%)	0	0	0	0	0	1 (7.7%)	0

Symptoms.—The typical illness with a history of acute appendicitis with the history of cramping epigastric or umbilical pain followed by nausea, vomiting, and localization of a dull aching pain in the right lower quadrant of the abdomen is easily diagnosed. However, many cases present atypical clinical pictures, and these, consequently, often are not recognized until perforation and peritonitis have occurred. In the experience of Eliason and

Johnson about 25 per cent of the cases of appendicitis were atypical in that one or more of the so called "cardinal findings" of acute appendicitis were absent.

TABLE VIII

Pathologic Type of Appendicitis	Total No. of Cases	Mortality
(1) Acute gangrenous	71 (31.7%)	13 (46.4%)
(2) Acute diffuse	42 (18.8%)	1 (3.5%)
(3) ("Just") Acute appendicitis	68 (30.3%)	5 (17.8%)
(4) No report given (no specimen)	28 (11.1%)	6 (21.4%)
(5) Acute ulcerative	10 (4.4%)	0
(6) Acute exacerbation of chronic	24 (10.6%)	2 (7.1%)
(7) Acute purulent	2 (0.9%)	0
(8) Subacute	5 (2.2%)	1 (3.5%)
(9) Chronic necrotic	1 (0.4%)	0
(10) Acute hemorrhagic	1 (0.4%)	0
Totals	252 (100%)	28 (100%)

In our series of acute perforated appendicitis only 40.7 per cent gave a history of cramping epigastric or umbilical pain, nausea, vomiting and localization of the pain to the lower right quadrant. This figure is considerably lower than those reported for acute unruptured appendicitis. Thus it is evident that the number of atypical cases is much greater in perforated appendicitis. Our attention must therefore be turned to a study of these atypical cases which comprise 59.3 per cent of the total in an effort to learn how better to diagnose them.

Constipation is frequently associated with appendicitis. However, enteritis with diarrhea may clear up but still leave an acute inflammation of the appendix. Diarrhea occurred in 6.25 per cent of the series and in 9.07 per cent of those cases complicated by general peritonitis. Seventy-five per cent of the cases with diarrhea occurred in children under ten years of age. The diarrhea was described as from two to 20 green, watery, foul stools daily. One patient with general peritonitis gave a history of a frequent urge to defecate without ability to do so. The presence of diarrhea delayed the diagnosis and operation from one to seven days. Initial back pain occurred in one instance in the absence of umbilical or epigastric pain. Nausea, vomiting and localization of pain to the lower right quadrant followed. At operation, a ruptured appendix with generalized peritonitis was found. This patient expired four days after operation with a paralytic ileus and myocardial failure. Relatively mild chills occurred in only a small number of the acute gangrenous appendicitis cases. Severe chills or rigors, associated with jaundice, occurred in two cases, both with pyelophlebitis.

Only 67 of the 252 cases (26.0 per cent) gave a history of one or more

previous attacks of appendicitis, the distribution in the four groups being approximately equal. It is noted that these cases were liable to early perforation, frequently in less than 24 hours. Perforation of the appendix was found to have occurred within 24 hours or less from the onset of symptoms in one-third of all those cases with a history of previous attacks. Three cases had perforated within six, eight and ten hours respectively. The present illness of those cases perforating before 24 hours was characterized by a more stormy course with severe sudden epigastric or umbilical pain, nausea, vomiting, mild chilly sensations, and localization of the pain in the right lower quadrant. Sudden cessation of the pain was noted in only a few instances.

The average duration of symptoms before operation was 2.5 days for those cases with general peritonitis, 2.8 days for those with local peritonitis, 11.6 days for those with abscess and 6.56 for those with abscess and general peritonitis. It is interesting to note that the average duration of symptoms in both the local and general peritonitis cases is practically the same. This suggests that the type and virulence of the invading organisms and the resistance of the host are important factors determining whether the peritonitis is confined to the right iliac fossa, or becomes quickly disseminated.

Signs.—Right lower quadrant tenderness was the most constant finding being definitely elicited at the time of admission in 70 per cent of the total number of cases. Complete absence of all signs was recorded in only one instance, one of appendiceal abscess. Suprapubic tenderness was noted in two cases of appendicitis with pelvic abscess. A limping gait was the symptom causing one patient to seek medical advice in one instance of peri-appendiceal retroperitoneal abscess of one month's duration. Jaundice occurred in only two cases, both having pyelophlebitis with multiple liver abscesses.

The average admission temperature for the abscess, local peritonitis, abscess plus generalized peritonitis, and general peritonitis groups are 99.9°, 101.1°, and 100° and 101.2° F. respectively. Higher temperatures up to 104 occurred frequently in general peritonitis, and to a lesser extent in appendiceal abscess. The mean temperature corresponds in all instances with the average temperature.

Laboratory Data.—Urine: Almost one-half (42.2 per cent) of all the cases of acute perforated appendicitis showed an albuminuria. The toxemia, resulting from the appendicitis and peritonitis and causing cloudy swelling of the kidneys, appears to be the explanation for these urinary findings. However, actual infection of the urinary tract may play a definite rôle in many of these cases.

In one instance, the patient, a young girl, was known to have had a definite pyelitis of long standing. She was submitted to the hospital for what was thought to be an acute exacerbation of this condition. A careful history revealed that the attack was ushered in by epigastric cramps, nausea, and

localization of the pain to the right lower quadrant and flank. Although the patient had urinary frequency and burning, and although the urine was filled with clumped pus cells, a diagnosis of acute appendicitis was made. Immediate operation revealed a perforated gangrenous appendicitis with local peritonitis.

The urinary findings in these cases usually cleared up within three to seven days after operation.

The average white blood counts in the four groups were as follows: (1) Local peritonitis, 17,000; (2) general peritonitis, 18,150; (3) abscess, 16,600; (4) abscess and general peritonitis, 14,462. In the general peritonitis group, the lowest white blood count was 10,550 and the highest 53,600. In the local peritonitis the lowest was 8,300 and the highest 30,000. In the abscess group, the lowest was 8,400 and the highest 44,000.

The average differential white blood counts were approximately the same, showing 83.1 per cent polymorphonuclear leukocytes in the abscess cases, 84.4 per cent in the local peritonitis cases, and 85.7 per cent in the general peritonitis cases. The highest individual percentage of polymorphonuclear leukocytes was 98 per cent occurring in a patient with general peritonitis.

Time of Operation.—There has been considerable controversy among surgeons as to the proper time to operate in acute perforated appendicitis. It is uniformly agreed that all early cases should undergo immediate operation. Concerning those patients who enter the hospital three, four or more days after the onset of symptoms, the disagreement arises as to whether immediate operation should be performed or Ochsner's conservative treatment instituted. It has been the rule in our clinic to operate immediately after the diagnosis of acute appendicitis has been made regardless of the duration of symptoms unless the patient is practically moribund.

Preoperative Treatment.—Preoperatively, if the patient shows signs of

TABLE IX

SHOWING THE RELATIONSHIP OF THE TYPE OF ANESTHETIC AND MORTALITY

Anesthetic	Local Peritonitis		General Peritonitis		Appendiceal Abscess		General Peritonitis Abscess		Total No. of	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
Spinal	6	0	9	1 (11.0%)	12	0	0	0	27 (10.8%)	1 (3.7%)
Ether and ethylene	36	0	33	7 (21.2%)	84	4 (4.7%)	10	6 (60.0%)	163 (65.2%)	17 (10.3%)
Straight ether	8	0	17	5 (29.4%)	18	1 (5.5%)	4	1 (25.0%)	47	7 (15.0%)
Nitrous oxide and ether	2	0	2	1	2	0	1	0	7	1
Avertin and ethylene	2	0	1	0	0	0	0	0	4	0
Local	1	0	1	0	0	0	0	0	2	0
Totals	55	0	63	147	117	5	15	7	250	26

NOTE: Two cases of patients with general peritonitis in extremis were not operated upon. Both died.

toxemia or dehydration, he is given adequate intravenous (glucose-saline) fluids.

The types of anesthesia used are set forth in Table IX.

TABLE X

SHOWING THE RELATIONSHIP OF TYPE OF APPENDICECTOMY INCISION AND THE MORTALITY

Type of Incision	Local Peritonitis		Generalized Peritonitis		Appendiceal Abscess		General Peritonitis and Abscess		Total Cases	Total Mortality
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths		
Gridiron or McBurney	40 (72.7%)	0	41 (65.0%)	6 (15.0%)	85 (72.6%)	3 (3.5%)	9 (3.5%)	2 (22.0%)	175 (70.0%)	11 (6.2%)
Right rectus.....	13 (23.6%)	0	19 (30.1%)	6 (31.6%)	25 (21.3%)	1 (4.0%)	6 (40.0%)	5 (83.3%)	63 (25.2%)	12 (19.4%)
"Midline" suprapubic	2 (4.4%)	0	3 (4.7%)	2 (66.0%)	6 (5.1%)	1 (16.6%)	0	0	11 (4.4%)	3 (27.2%)
Herniotomy (mistaken diagnosis)	0	0	0	0	0	0	0	0	1 (.4%)	0
Totals.....	55	0	63	14	117	5	15	7	250 (100%)	26

NOTE: Two cases of general peritonitis in extremis were not operated upon.

The gridiron or McBurney incision was used in 70 per cent of the total number of cases, the right rectus in 25.2 per cent, and the midline suprapubic in 4.4 per cent. The mortality rate in all the cases operated upon through a McBurney incision was 6.2 per cent; that of those operated upon through a right rectus incision was three times as great or 19.4 per cent, while that of those who had the midline suprapubic incision was over four times as great, or 27.2 per cent. Although this is a rather small series of cases, this is nevertheless evidence showing a definite progressive increase in mortality as the location of the incision is moved from the McBurney site toward the midline. In the local peritonitis group, there were no fatalities regardless of the type of incision used. It would seem that the gridiron incision is the incision of choice in acute appendicitis, ruptured or unruptured, and at any age.

The following advantages of the McBurney incision have been advanced:

(1) Relatively simpler procedure requiring less time.
(2) The appendix or appendiceal abscess is usually easily accessible.
(3) Soiling of the small intestine and other parts of the peritoneal cavity is reduced to a minimum.
(4) Mortality is decidedly lower.
(5) Shorter period of hospitalization.
(6) Elimination of traumatizing effect of gauze packing on intestines and peritoneum with subsequent formation of adhesions.
(7) McBurney incisions, even with prolonged drainage, are less apt to allow the formation of postoperative ventral herniae.

The usual operative procedure in our clinic consisted in the removal of the appendix in every instance in which the patient's condition was felt to warrant it. The usual appendicectomy included ligation and inversion of the stump. The appendix was ligated only, and not inverted, in 31 cases: two of general peritonitis, 23 of abscess, four of local peritonitis and two of abscess and general peritonitis. The two cases of general peritonitis and two cases with abscess died.

The appendix was not removed in 19 cases: 17 of abscess with one death, and two of general peritonitis with two deaths. Of the surviving 16 abscess cases in which drainage only was done, four (25 per cent) had a subsequent acute attack of appendicitis proved by operation within one year. A fifth case, having right lower quadrant pain, was operated upon six months after his initial operation and an active "chronic" appendicitis was found. Thus the mortality rate in those patients in whom the appendix was not removed was 15.8 per cent compared to 9.99 per cent for those cases in which appendicectomy was performed. It may be assumed that the patients in whom the appendix was not removed were more ill than in that group in which it was removed.

Drainage.—Every case in this series was drained with one to three cigarette drains, usually through the original incision. In the more recent cases of general peritonitis, drainage was frequently effected through a right McBurney incision, a left lower quadrant stab wound or a suprapubic stab wound.

The Average Duration of Operation Was:
43.3 minutes for general peritonitis
47.8 minutes for local peritonitis
45.0 minutes for abscess
58.5 minutes for abscess and general peritonitis.

Postoperative Treatment.—After operation, immediate transfusion is performed on practically all patients with general peritonitis and on many of those with abscess. The tonus of the intestines is kept up with repeated interval doses of pituitrin or pitressin, hot abdominal stupes, or intravenous injections of hypertonic salt solution. If this is accomplished, the patient is allowed fluids by mouth. Liquids were withheld only in the instance of vomiting or an ileus.

Postoperatively, the patients receive on an average of 3,000 to 5,000 cc. of fluid by oral, intravenous, hypodermoclysis of proctoclysis administration. If there was a tendency to distention, the Rehfuss duodenal tube was used.

Postoperative Course.—The temperature usually rose on the first day to 101° F., and fell gradually to normal some time on the third day in the great majority of cases. After the third day, the temperature was of an irregular remittent type, rising every afternoon, with the daily peak falling gradually to normal, some time between the eighth and twenty-eighth day,

dependent upon the extent of the infection and the adequacy of the drainage. The cases of general peritonitis tended to run a more prolonged period of fever with higher peaks and lasting on an average of 12 to 20 days. The abscess cases, when well drained, usually ran a febrile course of six to 12 days. The local peritonitis cases usually were afebrile after the eighth or twelfth day. In the fatal cases of peritonitis, the temperature rapidly rose to a terminal level of 106° F. or more.

In the general peritonitis and abscess group, two types of temperature curves were found, depending upon whether a general peritonitis or abscess was found at the time of operation. In those patients in whom an abscess was found at operation and in whom general peritonitis later developed, the temperature was found to follow the usual curve, reaching normal on the third day. Thereafter, the fever became remittent with high peaks of 103° or more, gradually receding to normal in 12 to 20 days. In the fatal cases, the temperature continued to rise after the third day to a terminal level of 106° or more. In those patients in whom a general peritonitis was found at operation and in whom one or more intra-abdominal abscesses appeared later, the temperature curve followed the usual curve until the eighth to tenth day when it rose to 103° or more, heralding the presence of the abscess.

As a rule, the pulse curve followed that of the temperature. A pulse of

TABLE XI

SHOWING THE IMMEDIATE CAUSES OF DEATH IN THE FATAL CASES. PNEUMONIA, PARALYTIC ILEUS AND CIRCULATORY COLLAPSE COMPRISED 54 PER CENT OF THESE CAUSES OF DEATH

Immediate Cause of Death	General Peritonitis	Appendiceal Abscess	Appendiceal Abscess and General Peritonitis	Total No. of Deaths	Percentage of Total Mortality
(1) Pneumonia	4	0	1	5	18.0
(2) Ileus	2	0	3	5	18.0
(3) Myocardial failure	4	0	1	5	18.0
(4) Fulminating general peritonitis	3	0	0	3	10.7
(5) Pyelophlebitis	0	2	0	2	7.0
(6) Subphrenic abscess	1	0	0	1	3.5
(7) Septicemia	1	0	0	1	3.5
(8) P. O. Shock	0	0	1	1	3.5
(9) Intra-abdominal hemorrhage, 9 days postoperatively	1	0	0	1	3.5
(10) Mechanical intestinal obstruction	0	1	0	1	3.5
(11) Status thymicolymphaticus	0	1	0	1	3.5
(12) Ruptured wound	0	0	1	1	3.5
(13) Undetermined	0	1	0	1	3.5

140 or greater was a grave prognostic sign, reflecting the severity of the infection or some complication.

Postoperative Complications.—Paralytic ileus occurred in 14 cases; eight with general peritonitis; two with abscess; three with both general peritonitis and abscess, and one with local peritonitis. Nine of these patients died. The frequency and high mortality of this complication bespeak its gravity and the necessity for its prevention. Bronchopneumonia occurred in nine instances with five deaths. Five cases of general peritonitis developed subphrenic abscess, one of which died. Fecal fistulae developed in five patients, one of which died. Myocardial failure was responsible for three deaths in patients with generalized peritonitis. Pyelophlebitis with multiple liver abscesses occurred in two cases, both of whom died, the condition not being definitely recognized before the autopsy. Two cases were known to be complicated by septicemia and one of these recovered. Profound postoperative shock was described in one fatal case with general peritonitis following the appendiceal abscess.

Sequelae.—Postoperative ventral herniae developed in 4.6 per cent of the surviving general peritonitis cases 4.4 per cent of the surviving abscess cases; 1.8 per cent of the local peritonitis cases. This herniation occurred in a McBurney incision in each instance but one—a midline incision. It is to be remembered that drainage was used through all of these incisions. Only two cases of general peritonitis, one with a McBurney and one with a right rectus incision, developed acute mechanical intestinal obstruction. This occurred in each instance within one month after operation.

Period of Hospitalization.—The average period of hospitalization was 22.9 days for local peritonitis; 25.3 days for general peritonitis; 23.8 days for abscess; 26.4 days for general peritonitis with abscess. The duration of stay in the hospital ranged from 11 to 90 days.

SUMMARY.—The report of this series of 252 consecutive cases of perforated appendicitis shows a steadily declining death rate.

We believe that these improved results are due primarily to better pre- and postoperative handling of the cases. This includes:

(1) Preoperative intravenous fluids.
(2) Repeated blood transfusions.
(3) The use of the duodenal suction drainage tube.
(4) The administration of morphia and pitressin to keep up the intestinal tone.
(5) The maintenance of adequate postoperative fluid intake, either the continuous drip method or by frequent intravenous injections.
(6) Proper psychotherapy by all who come in contact with such potentially ill patients.

We also attribute this reduction in the death rate of cases of perforated

appendicitis to such factors as the routine use of the McBurney incision and to the employment of spinal anesthesia unless contradindicated.

Recently we have grouped as donors any of those patients who have recovered from peritonitis.

The average age of the group of patients with local peritonitis was 27 years while 40 years was the average age of the group who developed abscesses. From this, one might conclude that the Ochsner method of treatment, if it were used, would be safer in the older group of patients.

We have not used enterostomy because we believe that all the benefits accruing from its use may be obtained by duodenal suction.

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THE APPENDIX PROBLEM

A PERENNIAL CAUSE OF PREVENTABLE MORTALITY

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THIS year (1936) is the semicentennial of the epoch making paper presented by Reginald Fitz, in 1886, in which he reported the autopsy findings in 151 patients who died with abdominal pathology which originated in the vermiform appendix. It is also approximately the semicentennial of the first successful, preoperatively diagnosed, operation for appendicitis by Thomas G. Morton, in 1887, and in the same year a similar case by Sands. Many other investigators, even before the Christian Era and up to the present time, have made invaluable contributions towards solving the appendix problem. We who have inherited their achievements should be stimulated by their example to continuous efforts in solving the problem as a cause of death.

Mortality Statistics.—During May, June, July, August, and September, 1936, there was a mild epidemic of poliomyelitis in Alabama and in some other states, during which 339 cases occurred in Alabama, 26 of whom died. As soon as an unusual number of cases were reported to the Alabama State Health Department, active steps were taken by the department to prevent the spread of the infection. The city and county health authorities and the Department of Health of the United States Government cooperated in every way in caring for those who were afflicted with the disease and in preventing its further spread. The press, civic clubs, churches, schools, and all other organizations, and the people generally, cooperated with the health authorities in preventing the occurrence of the disease. Those least informed in Alabama could not have been ignorant of the fact that there was an epidemic of poliomyelitis in the state and elsewhere. It occurred to the writer that if, in a degree, such coöperation could be secured in stamping out or preventing all preventable diseases and possible preventable deaths, such cooperative effort would be the cause of the saving of thousands of lives that are lost annually. Information secured from the Department of Public Health of Alabama gave the following mortality for appendicitis for the same months in 1935 as those during which the poliomyelitis epidemic had occurred in 1936: May, 24; June, 21; July, 32; August, 30; and September, 26 deaths, a total of 133 appendicitis deaths for the five months in 1935 or 26.6 per month. The Bureau of Vital Statistics of Alabama stated that the deaths due to appendicitis for the same months in 1936 "probably would not differ materially from that in the preceding year." The writer was amazed to find that so large a number die monthly in Alabama from the complications of the neglected appendix cases. It occurred to him, in the language of Doctor

Finney, Sr., "that something should be done about it," namely, these perennial deaths which are due to appendicitis.

According to the Statistical Bulletin of the Metropolitan Life Insurance Company for November 12, 1936, the death rate from appendicitis was 11.4 per cent per 100,000 for 1935, or 1.7 per cent lower than for 1934. However, the Bureau of Census of the U. S. Department of Commerce gives a mortality of 14.3 per cent per 100,000 population for 1934. It is interesting to note that the mortality per 100,000 population due to appendicitis from 1900 to 1934 has never been higher than 15.3 per cent per 100,000 population. In 1900 it was 9.7 per cent and rose gradually with recessions and fluctuations each year until the highest, 15.3 per cent, was reached in 1928. It was from 1925 until 1932 that it varied from 15 to 15.3 per cent (1931). For the years 1932, 1933, 1934, it was respectively 14.2, 14.1, and 14.3 per cent. There were 18,129 deaths in the United States in 1934 due to appendicitis. The mortality due to this condition in Chicago, Detroit, Los Angeles, New York City and Philadelphia averaged 13.7 per cent per 100,000 for 1934, whereas for 1935 it was 12.4 per cent, a reduction of 1.3 per cent, with Philadelphia having the lowest, *i.e.*, 10.9 per cent. The death rate of 60 selected cities in the United States for the year 1934 was 15.3 per cent per 100,000, whereas for 1935 it was 13.6 per cent per 100,000, a difference of 1.7 per cent. So, according to the statistics of the Metropolitan Life Insurance Company and the official reports of the Bureau of Census of the United States Government, the mortality due to appendicitis is being markedly reduced.

Appendicitis is not the only possible preventable cause of mortality. Table I, according to the Bureau of Census of the U. S. Department of Commerce, shows the statistical records of some such diseases for 1934:

The appendix is by nature a deficient, ill constructed test tube or cesspool of the intestinal tract. Anatomically, physically, and physiologically, it is a potentially obstructed tube with a terminal blood supply, the ingress and egress of its contents opposing each other. In the small lumen of the tube the opposing currents produce sedimentation and lamination which result in fecoliths in 59 per cent of the appendices as found in autopsies by Fitz. Intraluminal pressure produces intramural pressure and possible strangulation and consequent gangrene followed by rupture. There may be infection in cases where there is injury to the mucosa. There may be a hematogenous infection. Adolph Volz, in 1846, called attention to appendiceal concretions and their relation to perforations of the appendix. Von Zwalenburg, in 1904, and Wilkie, in 1914, reiterated faith in the belief that obstruction was a factor of major consequence for the occurrence of appendicitis. Wangensteen has recently emphasized the defective mechanism of the vermiform appendix—a potentially closed loop—and has designated it by nature as the sword of Damocles which always holds out a threat to the individual who has not had the appendix removed. According to Royster, at least three cases of prenatal appendicitis have been recorded. Cases also occur in the

extremes of life—in infancy and the very old. The appendix problem exists before birth and persists throughout life, unless or until it is removed. It is a little potential assassin (Royster) that may suddenly cause the most serious abdominal pathology and destroy the life of the individual. It is truly the sword of Damocles as far as the individual is concerned.

TABLE I

STATISTICAL RECORDS OF DEATHS FROM SOME DISEASES OTHER THAN APPENDICITIS DURING 1934

Cause of Death	Number of Deaths	Rate per 100,000 Estimated Population
(1) Infectious and parasitic diseases	148,124	117.2
(2) Cancers and other tumors	140,771	111.3
(a) Of the digestive tract and peritoneum	65,476	51.8
(3) Rheumatic diseases, nutritional diseases, diseases of the endocrine glands, and other general diseases	42,568	33.7
(4) Diseases of the blood and blood-making organs	10,250	8.1
(5) Chronic poisonings and intoxications	3,921	3.1
(6) Diseases of the nervous system and of the organs of special sense	134,365	106.3
(7) Diseases of the circulatory system	333,296	263.6
(8) Diseases of the respiratory system	114,879	90.9
(a) Bronchopenumonia (including capillary bronchitis)	41,923	33.2
(b) Lobar pneumonia	54,794	43.3
(9) Diseases of the digestive system	95,961	75.9
(a) Diarrhea and enteritis (under 2 years of age)	17,019	13.5
(b) Diarrhea and enteritis (2 years and over)	6,192	4.9
(10) Diseases of the genito-urinary system	125,171	99.0
(11) Diseases of pregnancy, childbirth, and the puerperal state	12,859	10.2
(12) Diseases of the skin and cellular tissue	2,144	1.7
(13) Violent and accidental deaths	132,022	104.4
(a) Suicide	18,828	14.9
(b) Homicide	12,055	9.5
(c) Accidental, other, or undefined	101,139	80.0
(1) By fall	20,762	16.4
(14) Automobile accidents (primary)	33,980	26.9

Appendicitis, A Neglected Medical Educational Problem.—In an outstanding recent book (1936) on the practice of medicine by a great medical man, appendicitis does not appear in the index of diseases in the text. Recently, in discussing the advances in gastro-enterology, an outstanding and learned member of this specialty left the appendix out of consideration entirely. Said he: "I shall actively oppose narrow specialism in any branch of medicine; there can be no doubt that such an unfortunate state has arisen and continues to exist, to the inevitable detriment not only to specialism but also to medicine as a whole. I prefer to be considered a properly trained internist who is devoting himself especially to diseases of the abdomen having

both direct and indirect relationship with the digestive organs. The men working in the domain of diseases of the digestive tract are not confining their efforts to a restricted field but are continually reaching out to explore all the areas that may conceivably influence that realm. They are constantly evolving new and useful ideas as to etiology, pathology, symptomatology, diagnosis and therapy in every ramification of the digestive system, every factor bearing on the digestive tract has been considered, every phase of its manifold functions has been studied; and this great volume of effort furnishes us with not only many stimulating problems but also with clues for their final solution. All signs, indeed, point to ever higher pinnacles of achievement. The fellow human beings who place their health, and thus their lives, in our hands, have the right to expect from us all the security that our high standards can provide." Pendergrass, in a paper on "The Small Intestine," omitted the appendix from consideration.

The appendix is evidently considered an entity, and the fact that it is a part of the intestinal tract and is so frequently the entire cause of serious disturbances in the entire digestive system and is the cause of so many preventable deaths seems not to be considered by many members of the profession whose active and helpful cooperation must be had in preventing deaths due to appendicitis.

Education, Experience and Ability of the Surgeon in Relation to Mortality.—Battle, in 1932, called attention to the large number of cases of appendicitis admitted to the hospitals of London. He said from the year 1873 to 1893, from one to 28 cases with inflammatory masses in the iliac fossa, with rarely more than 13 cases for any one year, were admitted to St. Thomas Hospital. All were turned over to the Medical Service. They rarely required operation and then only a simple incision and drainage. From 1869 to 1892, from one to 13 cases of peritonitis were admitted annually. "There was a change in 1893," said he, "in the character of the disease." In 1895, there were 59 cases admitted. This number increased each year until in 1913 there were 571 cases. The total admissions to St. Thomas from 1894 to 1930, excluding the war years, was 9,481. "The figures can only be comparatively correct," said Battle, "for in more recent years a new section of abdominal pain has appeared." I have summarized his report because to my mind it represents an epoch in medical education at least as to the recognition of abdominal pathology due to the neglected appendix or possibly in some cases a recognition of early cases of acute appendicitis.

The change in 1893 in the character of the disease—appendicitis—as noted by Battle, which was doubtless not a change in the disease but a change in the educational status of the men who handled the cases, was also noted before this time in America. Following the appendicectomies by Morton and Sands in 1887, American surgeons recognized immediately that a change in the recognition and treatment of the disease was necessary. Many made early and valuable contributions on the subject of appendicitis and its complications. In 1890, the subject of the treatment of general, septic peritonitis

was discussed at a meeting of the Southern Surgical Association. In discussing this paper, Dr. W. E. B. Davis said, "These cases are before us constantly. The physician should be educated up to the point to recognize this condition so that he may call a surgeon at a time when he can do some good and not after it is too late. We are losing patients by delaying operation and simply because the general practitioner does not appreciate the importance of the disease. We are rarely ever called until three days after rupture has taken place. Before this time, all manner of treatment has been resorted to and we are now called in and expected to operate. I have suggested an operation which, I believe, is practicable in cases of suppurative peritonitis with great tympanites and obstructive symptoms and it is this: Open the abdomen, incise the intestine, wash out with hot water and make an artificial anus. This will relieve the tympanites and give the patient the best chance to recover." Dr. J. D. S. Davis said: "In those cases of appendicitis where there is a formation of pus, the abscess should be opened. We do a life-saving operation. We should not subject the patient to too great a shock in removing the appendix but after the patient recovers from the appendiceal abscess, we can then remove the appendix." That was and is modern surgery as we know it today. The question of purges or salines in cases of appendicitis or its complications up to 1893, and even later, was one that caused considerable discussion among surgeons and medical men. Dr. Joseph Price said, in 1893, "It is curious how medical men differ from us. I heard Professor Hare say that under no circumstances would he give salines in cases of appendicitis; that surgery should precede the saline treatment." Dr. Hunter McGuire, in 1893, said, "Not more than a year ago in discussing the subject of appendicitis, I said, 'there was no subject in surgery about which I was more uncertain than when to wait or when to operate in these cases.' I confess," said he, "in these cases, I have never operated too soon but have many times operated too late." Doctor Tiffany of Baltimore said, "The majority of cases of appendicitis we do not see at all. An early diagnosis makes a clean operation, a clean belly cavity and the patient does well." Dr. Bacon Saunders, in 1893, discussed an extraperitoneal method of operating in "Certain Cases of Appendicitis" and urged that all intra-abdominal abscesses be opened extraperitoneally.

Sir James Berry, in 1932, called attention to his letter to The Lancet in 1889 in which he reported 41 abdominal abscess cases opened by incision with only two deaths (extraperitoneal incision), and 39 cases treated by free incision into the general peritoneal cavity in which there were 32 deaths. He referred to the free incision into the general peritoneal cavity in which there were 32 deaths in 39 cases as an operation which was then beginning to become fashionable and which is now—1932—so common. "I do not claim," said he, "that the two classes of cases are in every respect strongly comparable but approximately they are representative of the two widely different methods of treating appendicitis by two sets of surgeons in the same hospital in the '90's, and also at the present time (1932)."

He also, in 1932, referred to two historic cases as showing the manner in which similar cases were handled in 1902 and 1925 by different surgeons as indicating the difference in the methods of operative treatment:

Case 1.—King Edward VII. Age 61. 1902.

Saturday, June 14	Abdominal discomfort.
Sunday, June 15	5 A.M.: Seen by doctor for acute attack of pain. Afternoon: Seen by consulting physician. ? rigor.
Wednesday, June 18	First seen by surgeon. Temperature. Pain and swelling in the right iliac fossa. Diagnosis: "peri-typhlitis" (i.e., appendicitis).
Thursday, June 19 Friday, June 20	Above signs disappearing.
Saturday, June 21	Seen again by surgeon. Swelling nearly gone. Temperature normal.
Monday, June 23	Seen by surgeon.
Tuesday, June 24	Operation. Limited incision into intraperitoneal abscess. Much foul pus drained. Appendix not removed, not seen, not felt.
Afterwards	No recurrence. Lived nearly eight years.

Case 2.—President Ebert of Germany. Age 54. 1925.

Sunday, February 22	Very well all day.
Monday, February 23	In night slight abdominal pain. 9 A.M.: Seen by doctor. 5:30 P.M.: Severe abdominal pain. Doctor sent for. 6 P.M.: Tenderness and slight rigidity in right iliac fossa. 9 P.M.: First seen by surgeon. Immediate operation to be performed. Moved to nursing home.
Tuesday, February 24	12 Midnight: Operation. Free incision into peritoneal cavity. Removal of appendix. Turbid nonodorous fluid. Intestines distended. Pelvis quite free from fluid.
Saturday, February 28	Death. Postmortem examination.

He said with reference to King Edward's case, "The incision was made low down until an abscess was reached and opened; tubes and gauze were inserted and nothing more done." "There is no little irony," said he, "in the fact that the popularity of the modern operation of treating appendicitis by free incision and removal of the appendix and abscess without regard to limiting adhesions is largely based upon ignorance of what was really done at this famous operation." In discussing the case of President Ebert, he said, "Twenty-three years have elapsed. Surgical fashions have changed. The septic focus—this dangerous appendix—must be removed at the earliest opportunity at any cost. Surgical technic has greatly improved, it is true, but the disease called appendicitis remains what it has always been even when it was not known by that name. As I read this report (the Ebert case) as given, I could not help feeling that I was back again in the good old days at St. Bartholomew in the '90's when I, as surgical registrar, was myself seeing things then and recording them in the postmortem book and in my annual surgical reports."

It is not stated that a diagnosis in King Edward's case was made until the surgeon saw him on the fourth day after the initial attack. The "swelling

in the right iliac fossa" had formed during the four days. The indicated surgery was correctly performed at the proper time. If the diagnosis in President Ebert's case could have been made early on the morning following the attack during the night, and the operation performed early or within a few hours, all can agree it would have given him a better chance for recovery. Surgeons have not and possibly never will be able preoperatively to determine the absolute state of pathology in or due to an infected appendix and the course the disease will pursue, but surgeons do know that the infected appendix is treacherous and deadly and the safe thing to do is to remove it unless an abscess is formed which can be opened extraperineally, or the patient is growing progressively better. It appears to the writer that neither case was a good surgical risk. They correspond in type with the two cases that died in the group of 2,106 cases in Finney's series. Finney said, "These deaths were in men of middle age, of short, thick pudgy build, marked beforehand as relatively poor operative risks and both of whom were subjected to through abdominal exploration as well as the appendicectomy through a right rectus incision. Both followed the same postoperative course; a paralytic ileus with progressive distention and total lack of peristalsis, dying after about a week with a terminal pneumonia. In each case, the absence of peritonitis was definitely proven."

Boland, in 1932, in a review of 4,272 cases of acute appendicitis in Atlanta, called attention to the large number of articles on the subject of appendicitis which had been published and which occur annually in medical and surgical journals. With irony he called the attention of the Section on Surgery, General and Abdominal, of the American Medical Association to the fact that only five papers had been read in the Section in the past 15 years and that the last one was read five years prior to his paper in 1932. "The medical profession," said he, "must not let familiarity with the commonest of all surgical diseases induce criminal somnolence."

Haggard says the mortality of appendicitis decreases with the experience of the surgeon. Bowers called attention to the difference in mortality of surgeons. In the cases reviewed by him, one surgeon had a mortality of 20 per cent, another 3.3 per cent, while several had no mortality. In 2,806 cases of appendicitis reviewed and reported by Reid, 72 different surgeons operated; "the mortality of the individual surgeons varied greatly."

Education of the Public as to Appendicitis.—The appendix problem has been, is, and should continue to be an educational problem. The solution of the problem depends upon certain very definite information which the people, individually and as a whole, must have relative to the symptoms and physical signs of appendicitis. It is the writer's conviction that the educational campaign for the purpose of giving this information to the people should be adequate, continuous, and orderly, and should be sponsored by the Health Department of the National Government, and of the states, counties, and cities, and that the instruction should extend to and be a part of the instruction of children in all grades of schools, so that the child, at least of

school age, can protect itself in case of an illness with abdominal symptoms due to appendicitis, or possible appendicitis, until he has been seen by a competent physician or surgeon.

If every case of appendicitis could be diagnosed early and operated upon promptly, it would be of inestimable value socially and economically, and would relieve patients of untold suffering and agony and would reduce the mortality from this disease to an incredibly low figure. The children in the schools, the boys and girls and young men and women in colleges and universities, and the people generally, should be told the importance of the alimentary tract and something of its structure and function, and in this instruction the appendix should not be left out of consideration. But it should be especially emphasized that it is frequently the cause of a serious illness (appendicitis) which, if not recognized early and handled properly from the beginning, may result fatally. It should be generally known that the first symptom of appendicitis is epigastric pain or distress which is usually followed by nausea and vomiting and later pain on pressure in the lower right side, followed possibly by temperature, and that purges and opiates should not be given in such cases. Individuals and the people generally should know the importance of the timely advice of a good and properly prepared physician in such cases. It should be known by properly prepared physicians that purges and opiates in such cases must not be given until the appendix is absolutely eliminated as the cause of the illness. It should be known by all that an early diagnosis should be made and that an early operation is a safe surgical procedure followed by a very low mortality, less than one-half of one per cent, and yet if complications develop from the neglected attack, there may be a very high mortality rate.

McClure, Reed, Boland, Roberson, Poole and others have called attention to the fact that certain groups under proper control and regulation have a very low mortality due to appendicitis. In 300 cases of college students reported by Roberson, there was only one death, and in 219 cases of college students and nurses reported by Boland, there were no deaths. Horace Reed says, "In our own state efforts are being made to educate the public on the early signs of acute appendicitis. In one large organization in our state in which there are something like 4000 employees this information has been disseminated and over a period of nearly three years there has not been a death from acute appendicitis." Poole referred to the fact that patients are now admitted earlier to the Employees' Hospital of the Tennessee Coal, Iron & R. R. Co., Fairfield, because the industries of the company are more centrally located and the hospital is more accessible to the employees. It has been observed by McClure, with whom I agree, that nurses with appendicitis are promptly diagnosed and operated upon with practically no mortality.

Important methods are being used in certain cities and states to reduce the mortality due to appendicitis, and the efforts have met with considerable success, but not the success it would appear that should be expected. Notwithstanding this, great good has been accomplished and will be accom-

plished, and those who started the campaign in Philadelphia, and especially Doctor Bowers and his associates and the health authorities, cannot be commended too highly for the work which they have done. It is really outstanding. What they have done and what has been accomplished show how difficult it is to handle the appendicitis problem. It is a complex, compound problem. It is a problem that vitally concerns the people as a whole and every individual who still has his appendix within his abdomen. It is known that it is difficult to disseminate knowledge and difficult to get people and individuals to realize that they do not know all about a problem. Even after the people have the knowledge that they should have with reference to appendicitis, there will be even then a large number of insidious, deceptive cases that will slip up on, and take the life of, an individual or individuals under the guise of an illness with comparatively slight symptoms of the destructive pathology in the abdomen which threatens, and will destroy the life of, the individual unless the diagnosis is made and the appendix is removed. The most educated and informed citizens, and even physicians and surgeons, can be and many are attacked by this insidious disease, and the disease progresses to such a stage that serious complications develop even before the individual recognizes that he is seriously ill. The appendix is truly, as Royster says, an assassin.

Diagnosis.—The insidious and deceptive nature of an attack of appendicitis with the variable symptoms and signs that may be present may cause the most learned and experienced physician or surgeon to miss a correct diagnosis. The attack may mimic in its symptoms, physical signs and laboratory findings, many diseases in the abdomen and remote therefrom, or there may be a most virulent infection of the appendix with practically no symptoms indicating its diseased condition. It may simulate many different diseases and many diseases may simulate it. There are some members of the profession who can paint the picture of the preoperative diagnosis of appendicitis so plainly as to cause one to see the appendix present itself before the incision is made, and after making the incision the appendix is found and removed with the greatest difficulty. The diagnosis may be comparatively easy. There are cases, however, that are most difficult and require thorough study. In such cases all means of diagnosis should be used. There should be no delay, but there should be a thorough study of the case which will enable one to make a correct diagnosis or at least to decide that the operation is or is not indicated. The patient should be so studied as not to permit the disease to become progressively worse from the appendix or any other possible intra-abdominal cause amenable to surgery. In this study there should be:

First, a complete, accurate and correct history of the case and especially of the present illness. Second, a complete and thorough physical examination. Third, a total and differential blood count and complete examination of urine. Fourth, the usual symptoms and physical signs of appendicitis must be properly correlated and interpreted. Fifth, in certain cases even of the acute type a diagnosis can be confirmed or corrected by a cystoscopic

examination and roentgenologic findings. Sixth, it is most important that pneumonia be eliminated as the cause of the abdominal symptoms.

In a large series of cases, Livingston found hyperesthesia present in the appendiceal triangle in 86 per cent, leukocytosis in 76 per cent, pain in 75 per cent, nausea and vomiting in 70 per cent, temperature in 67 per cent, rigidity in 59 per cent. Reid found in 1,465 cases of acute appendicitis, the following clinical data: Nausea 78 per cent, vomiting 73 per cent, abdominal pain 94 per cent, rectal tenderness 44 per cent, purgatives 36 per cent, and a typical history in 53 per cent. Pain on pressure over the appendix was present in nearly 100 per cent. He considers this the most important single symptom, and wisely says, "When this tenderness is present, the burden of proof is certainly on the examiner to show that it is not due to appendicitis." Muscle spasm and rigidity were usually but not always present. In some cases pain was elicited only on very deep pressure; in these cases the appendix was usually behind the cecum or in the pelvis. Nausea and vomiting usually occurred in the first few hours of the disease, but in some cases it did not appear until late. Tenderness, induration or a mass on rectal or pelvic examination was present in almost half of the cases. "Rectal examination is of particular value in children in whom a diagnosis is frequently very difficult." Reid's cases were admitted late—unruptured cases averaged 44 hours.

George Robertson published a most important paper on "Disturbed Reflexes; Their Significance in Acute Abdominal Diseases," and states that "absolute uniformity of symptoms in appendicitis is not found." He refers to Cope's division of the abdominal cavity into "the demonstrative area" and "the silent area" as being good. "Cope defines the 'demonstrative area' as being the whole peritoneal lining of the abdominal cavity, minus the pelvis and central and inferior parts of the posterior abdominal wall which is designated as the 'silent area.' In a large number of cases studied, Robertson found that rigidity in appendicitis does not occur unless the infected appendix is adherent to the 'demonstrative area,' or adherent to the omentum or intestine or a viscus which is involved in the infection and is itself adherent to the parietal abdominal wall, or the appendix is ruptured and the parietal wall is contaminated by its contents." From my own cases and the observation of a large number of others, I agree with Robertson that if rigidity occurs in appendicitis, it indicates an involvement of the peritoneum in the infectious process. Rigidity may be an early or a very late finding. The diagnosis, if possible, should not be delayed until the infectious process has involved the parietal peritoneum, thus producing rigidity. Robertson designates rigidity as a "sensori motor reflex." He states that "strong sympathetic stimuli from a diseased viscus are not responded by the contraction of a voluntary muscle. They are responded by a strong and continued reflex contraction of the plain muscles of the gut. This is the true viscero-motor reflex. It is not an objective sign but is a subjective symptom, and is expressed by the patient as pain of a certain character and frequently located far away

from the diseased viscus. The epigastric pain of acute appendicitis is due to a spasmodic contraction of the pyloric sphincter, and is described by the patient as epigastric. The periumbilical pain of acute appendicitis is due to strong peristaltic contraction of the lower ileum against a contracted ileocecal sphincter and is described by the patient as periumbilical. This reflexed disturbance is visceral and is of the autonomic nervous system. Visceral abdominal pain is due to vigorous peristalsis of involuntary muscle or contraction of sphincteric areas often at a distance from the point of stimulation. Rigidity on the other hand may be general or local, depending upon the extent of involvement of the parietal peritoneum. It is a sensori motor reflex."

Murphy said that the primary pain with a free appendix is always referred to the epigastrium. The secondary pain is usually not colicky, but of the typical inflammatory type and is due to periappendicitis. Rutherford Morrison said, "There would be no percentage of deaths from appendicitis if every case commencing with acute pain and developing tenderness and rigidity of the abdomen and quickening of the pulse were operated upon within 12 hours." This statement with reference to pain and rigidity, as well as McBurney's point of greatest pain, is misleading. They are frequently quoted in the literature on appendicitis, but the use of the word "pain" is not explicit. To wait for subjective pain at McBurney's point is to court disaster. It must be recognized that in most cases, the primary or initial pain is epigastric, umbilical or a generalized abdominal pain. There is usually tenderness on pressure at McBurney's point or at the point where the appendix is in the beginning, but the subjective pain and objective rigidity at this point may be and most often are late developments and indicate complications.

Chronic Appendicitis.—Those who are interested in chronic appendicitis would do well to read carefully McClure's paper which was presented to this Association in 1930. Foy Roberson in 1934, also strikingly called attention to what he regards as being chronic appendicitis. Royster has studied the subject most thoroughly and in discussing Roberson's paper said, "Let me illustrate what he (Roberson) said about the disease. Why should we use the terms 'chronic' or 'acute'? The attack is not the disease, only a knock at the door saying 'Let me out.' The chronic type is sometimes difficult to diagnose but I stand pat on the idea that the fulminant case is only the accumulation of years of pathology."

Haggard says chronic appendicitis is so easy to say and yet means so little. "We have little reason for diagnosing chronic appendicitis in the absence of definite acute or subacute attacks." There were in his large series of operative cases only 113 chronic cases. The writer has operated upon only one patient with a diagnosis of chronic appendicitis, but he has operated upon patients with the symptoms and signs of acute appendicitis which the pathologist reported as chronic appendicitis. He believes, however, that there are many cases of chronic appendicitis which should be operated upon, but in his experience he has not been able in his own mind to insist upon an

operation in such cases. This does not alter his conviction, however, that chronic appendicitis is widely prevalent and that many chronic cases become fulminant, acute cases.

Illustrative Case Reports

Case 1.—In 1912 a doctor was operated upon by Doctors Davis and Talley. He had had for a number of years pain on pressure over McBurney's point, and for the last year or two before the operation, occasionally late at night, he would vomit what he had eaten at lunch and at dinner. There was no epigastric pain, no tenderness, no acceleration of the pulse. The only symptom present was pain on deep pressure at McBurney's point and vomiting. He knew, or believed firmly, that he had a chronic appendix and he expected to be operated upon at a convenient season before he had an acute attack. He does not remember ever having had a previous acute attack of appendicitis. The acute attack which occasioned the operation began at 1 A.M. after he had attended an elaborate dinner the night before. He vomited two or three times during the night, got up early and operated on several cases before lunch. At lunch he took broth and immediately became very nauseated. He left the table and went to bed. He was very sick with nausea and exhaustion during the entire afternoon and night. He took no medicine. At 10 P.M. he decided that he had an acute appendix, and he was operated upon the next morning at 10 o'clock. He knew that he should have been operated upon as soon as a diagnosis was made but he did not want to trouble anyone at night; the diagnosis was made late. Up to that time he thought it was an indiscretion in diet. At operation a right rectus incision was made and a very large, gangrenous appendix presented itself. It was removed and a very small drain was placed just within the peritoneum in the lower angle of the incision. There was an uneventful recovery. This doctor believes firmly that if he had not been operated upon at the time he was for the acute attack of chronic appendicitis, he would have died.

Case 2.—Another instance was that of a doctor whom I saw in an acute attack of appendicitis in 1926. Since it was a mild attack, he insisted that he could not take the time to be operated upon unless it was absolutely necessary. He improved steadily and within two or three days resumed his duties. In 1928 his wife called me late one night and asked me to come at once as her husband was very ill, and before I could get dressed she called again and said she was afraid he would die before I could get there. When I saw him he was in the greatest agony, vomiting, bowels moving, cold perspiration, slow pulse, generalized abdominal pain, which was most acute in the upper right quadrant. Since his mild attack of acute appendicitis in 1926, he had been troubled with gastric distress for which he took bicarbonate of soda. I had told him at different times since his acute attack that he had chronic appendicitis following the acute attack, which was the cause of all of his trouble. He believed it was due to his gallbladder. When I saw him in his latest acute attack, I believed firmly that all of his trouble was due to his appendix. I knew he would be operated upon and I gave him half a grain of morphine immediately and within 30 minutes another quarter of a grain, after which he was more or less relieved of his severe pain. I wanted him to go to the hospital that night but he delayed until early the next morning. Doctor Talley and I operated about 10 A.M. the following morning. He had a large, gangrenous, obstructed, unruptured appendix. The incision was closed without drainage and he made an uneventful recovery, but about a year later he had a large intramural abdominal abscess just above the old appendix scar.

The writer now has in his files 86 cases which he believes have chronic appendices. Some of them have subacute attacks periodically. He has warned all of them that they may have a fulminating attack at any time.

Case 3.—A patient on whom I operated was of considerable interest to me. A young woman, a school teacher, age 24, had been thoroughly examined by members of the staffs in two high class hospitals with no diagnosis of any abdominal pathology. She had for the past two years been suffering with indigestion (dyspepsia) and pain in the right lower quadrant and just above the symphysis, metrorrhagia and menorrhagia. The G. I. series and examination of the urinary tract were negative. She had been on a diet for several months. As she had been treated for the menstrual trouble over a long period of time by good men, I decided to perform a diagnostic curettage. Macroscopically there was no evidence of pathology in the uterine scrapings. Because of the indefinite pain in the right lower quadrant and just over the symphysis, although the pelvic examination was negative, I decided while the patient was under the anesthetic, to perform a laparotomy for the purpose of removing what I regarded as being a chronic appendix. A midline incision was made between the symphysis and the umbilicus. On examining the uterus, a rough surface was found on the posterior of the right cornu. Running from the omentum in the pelvis, there was a very sharp, fibrous cord, about 12 cm. in length, extending to the tip of the appendix. The appendix was long and there were several kinks in it. The appendicectomy was performed. There was an uneventful convalescence, and there have been no abdominal or menstrual symptoms since. There were definite indications for the above operation, and the result has been extremely gratifying to the young woman, her family and friends.

Preoperative Preparation.—It is recognized that in the absence of the very greatest emergency that every patient can be properly prepared for an operation. This may require the giving of glucose and normal salt solution intravenously, normal salt solution by hypodermoclysis or the transfusion of blood and such anodynes and other medication as may be indicated. The preoperative treatment may be instituted and carried out during a thorough study of the patient, during which time the patient may be gotten into the very best possible condition to undergo the operation. The operation can be performed when indicated but it should certainly not be performed until the patient has such preparation as will enable him or her to be in the best possible condition to undergo the operation; and if, under such treatment, the patient becomes progressively better, the treatment may be continued until it is indicated that the operation should or should not be performed.

Operative Procedure.—Desirable as it is to avoid controversial questions or problems, yet in matters that are fundamental, one who avoids such issues cannot be intellectually honest. The question of immediate operation after the indicated preparation of the patient or delayed and possibly nonoperative treatment of the patient with local, diffuse, spreading, or general peritonitis is one that such surgeons as Richardson, Ochsner, Murphy, the Mayos, Price, McMurtrey, Haggard, Sr., Sherren, the Davises, and many other pioneer surgeons considered most seriously. The same question or problem is now uppermost in the minds of surgeons who are striving to render the greatest possible service to their patients. It is only second in importance to an early diagnosis and early operation in which the mortality is practically nil. Almost perfect results are reported by some surgeons who practice either the immediate or delayed procedure in the indicated case. The surgeons who get such a low mortality by either procedure evidently have adapted the procedure

to the patient rather than the patient to any fixed plan of preparation and operation. The patient with an abdominal complication of appendiceal origin is individualized, studied, treated and operated upon immediately when indicated, or is not operated upon when not indicated. Each experienced surgeon uses the preoperative, operative and postoperative procedures indicated in the individual case that will give, according to his knowledge and experience, the patient the best chance to recover. The surgeon owes it to the patient to use at least the known and approved methods of preoperative, operative, and postoperative treatment, which when used by the ablest surgeons give good results.

The expression by Richardson that "there are instances in which we see the case too late for the early operation and too early for a safe late operation" frequently occurs in the literature. To understand what the author meant by such an expression, it would seem that it is only right and proper to consider the entire context from which the quotation is abstracted in order to understand the real meaning which the author wished to convey. For this reason, I am quoting the entire paragraph from Richardson's original paper, in 1894, in which the expression "it seems to me—though I am by no means convinced of the truth of this assertion—that there are instances in which we see the case too late for the early operation, and too early for a safe late operation" originally occurs:

Richardson said, "I have often seen a patient for the first time in the third, fourth, or fifth day of an attack of severe type in which Nature had succeeded in opposing an adhesive barrier to further extravasation. Under these conditions the most important and difficult question arises—whether to operate or not. I have considered this question many times. It is during these days—the third, fourth, and fifth, or later—that the early operation may be said in some cases to be too late. The extravasation from the perforated appendix has taken place; the harm from this extravasation has been done; Nature, in her own way, has successfully, thus far, opposed this extravasation. The adhesions are not strong, and in separating them we are almost sure to contaminate the rest of the peritoneal cavity. It seems to me—though I am by no means convinced of the truth of this assertion—that there are instances in which we see the case too late for the early operation, and too early for a safe late operation; that if we operate we undo the work that has thus far been successfully accomplished by Nature, and that we convert a case that is doing well into a case of fatal general peritonitis. This is one of the most important questions in connection with the discussion of this disease. I do not mean to assert that interference in a localized peritonitis on the third, fourth, or fifth day is inadvisable. I have operated many times at this period. I have done this, however, with the greatest care not to break down the recent adhesions. There is no more difficult operation in surgery than that of removing an appendix at this stage without infecting the general peritoneal cavity. I do not mean to assert that, on the third, fourth, or fifth day, in a case that is getting on well, with a localized abscess, we should delay; but

the reasons I have given must appeal to one who dreads the presence of infecting material in the peritoneal cavity. The objection to leaving to itself a case in which presumably the adhesions are not strong is the giving way of these barriers under pressure and consequent fatal extravasation. That this danger is by no means slight is seen in the constant occurrence of a general peritonitis in cases that are apparently doing well. Where the symptoms of general peritoneal infection appear suddenly, in the course of a localized peritonitis, several hours at least must elapse before the surgeon can attempt to repair the mischief. Where the adhesions are broken down by the operation these efforts to cleanse the peritoneal cavity can, of course, be made at once."

The conservative treatment of appendiceal peritonitis as advocated by A. J. Ochsner, and later by Sherren, and the application of Richardson's principles of operative treatment, were recently very lucidly discussed in a paper by Alton Ochsner. This paper gives valuable information relative to the deferred operation in the indicated case.

Abscesses.—It is very clear that Doctor Richardson believed that in cases seen late—on the third, fourth, fifth or sixth day and later—where an abscess was being formed, and the patient was doing well, there should be no interference—no operation until the abscess was firmly walled off and could be opened extraperitoneally without reinfecting the patient. This position relative to opening abscesses, so as not to contaminate the abdominal cavity, was taken by Robert James Graves, about 1840. "One of his original ideas was a mode of treating hepatic abscesses so as to insure the patient against the risks consequent on the escape of purulent matter into the cavity of the peritoneum. The case cited by Graves was that of an hepatic abscess. The surgeons who saw the patient felt that the signs were too diffuse and the situation of the abscess uncertain, so that the operation not only afforded little chance of success but might prove very detrimental. They accordingly refused to operate. It occurred to Graves that if an incision were made within one or two lines of the peritoneum and kept open with a packing, the abscess might tend toward and finally rupture through it. One of the surgeons, Mr. MacNamara, finally consented to perform the operation as described. The result was awaited by Graves with much anxiety; two days later, the patient sneezed and a large quantity of pus escaped, followed by rapid recovery of the patient. Graves and others used this method on additional cases with much success." The earlier operators were so impressed with the importance of avoiding a recontamination or reinfection of the peritoneal cavity that many of them advised a midabdominal incision in order to locate the abscess before attempting to drain it extraperitoneally. Of course, this was an extra hazard to the patient and in time this plan was abandoned, and it became almost universally recognized, as it is now, that abdominal abscesses should be opened extraperitoneally. The abscess should be opened, if possible, where it points, at the place of greatest pain or tenderness or fluctuation. Herrick and McDill, in operating upon liver abscesses,

emphasized the importance of avoiding contamination not only of the pleural cavity but also of the abdominal cavity. Elkins has emphasized this in operating for subdiaphragmatic abscesses. The Herrick and McDill operation of resecting a rib (the tenth), pushing up the pleura, incising the diaphragm and attaching the diaphragm to the intercostal muscles and opening the abscess, only if it was walled off from the peritoneal cavity, and in case it was not, packing the abscess area in the liver with gauze and opening it within 24 to 48 hours, avoids contamination of the pleura and abdominal cavity and gives a remarkably low mortality record in operating on abscesses of the liver. It is found in practical experience by good surgeons that most intra-abdominal abscesses can be opened without reinfecting the abdominal cavity. Where the abscess is inaccessible and must be opened intra-abdominally, it appears that it would be ideal to have the intra-abdominal drain so placed that the drainage tract would be walled off as in liver abscesses before the abscess is opened or ruptures spontaneously. It seems to be agreed that those patients who are seen early after an appendiceal rupture or intra-abdominal rupture of an appendiceal abscess should be operated on without delay. In the case, however, seen late, where there is no evidence of abscess formation and there has been ample time for Nature to have formed an abscess and the patient is desperately ill, the question is whether or not this patient should be operated upon immediately after proper preparation or should be delayed as to operative treatment until an abscess is formed. In such a patient, which method gives the better chance of recovery? Extreme positions have been taken with reference to this type of patient. There are those who believe that immediate operation should be performed. There are others who think that such a patient should be put upon treatment—Fowler-Ochsner-Murphy treatment—and that the operation should be performed only after the patient is in condition to withstand the operation or be operated upon successfully. All surgeons, I am sure, would adopt the latter procedure if they could know that such a procedure would be in the interest of the patient. Richardson, as I interpret what he said, used the latter method of treatment in those cases seen late that were improving.

In 1921, the writer in presenting a paper to this Association on "Conservative Surgery in Non-Malignant Diseases of the Pelvic Organs" stated that extreme positions have been taken and that there are doubtless good reasons for the extremes. While this is true, good judgment will always dictate the solid foundation or principle on which those who hold to either extreme may compromise in the interest of the general good—in this case, the patients. Ultraradicalism and ultraconservatism may be equally disastrous—the one precipitately, the other slowly but surely. He believes the same may be said with reference to the appendix.

Mortality as Reported by Surgeons.—In an editorial in the American Medical Journal, June 20, 1936, on "Reducing the Mortality of Acute Appendicitis," reference is made to the record of Dr. LaGrand Guerry's report of a mortality rate of 1.4 per cent in a group of 135 cases of gan-

grenous ruptured appendicitis with diffuse peritonitis treated by the Ochsner method, and also to 502 consecutive cases of acute appendicitis reported by Guy W. Horsley, Jr., in which an immediate operation was performed with a mortality rate of 0.8 per cent, with the remark wisely that "there can be no doubt that the skill and judgment of the surgeon play an important part in the mortality rate of any operative procedure." H. A. Gamble, has recently reported 129 operated cases of peritonitis due to appendicitis with two deaths, a mortality of 1.55 per cent.

Bowers has shown that the reduction in mortality due to appendicitis is a most difficult task. In his review and thorough analysis of 8,216 cases in the hospitals of Philadelphia after an intensive educational campaign, it is shown that there was a reduction of 1.16 per cent mortality in 3,095 cases for 1930 as compared with the mortality in 5,121 cases for 1928 and 1929, the mortality for the two series being respectively 4.81 and 5.97 per cent. The decrease in the time (11.81 hours) of admission in the 1930 series and its relation to decrease in perforations—from 42.73 to 35.45 per cent—together with the relation of purgatives to spreading peritonitis, show clearly the way to prevent mortality due to appendicitis. J. M. T. Finney, Jr., in a study of 3,913 cases, and many other surgeons who have studied and reported large series of cases have shown that the mortality in simple uncomplicated appendicitis is very low—less than 1 per cent. Alton Ochsner, McClure, Gile and Bowler, Coller and Potter, Garlock, Boland, Maes, Reid, Keyes, Haggard, Roberson, and many others in this and other countries have recently presented notable papers on appendicitis reviewing large series of cases. Each one has emphasized the type of case in which mortality occurs, and the way to prevent or reduce the mortality. In studying their papers, it is not always clear that surgeons understand each other in discussing the different complications of appendicitis, and there is a great difference in mortality in certain types of cases as given by different surgeons; but the average mortality of the whole series of each good surgeon when compared with that of other good surgeons does not differ markedly. All surgeons agree that the cases of appendicitis in which the infection extends beyond the appendix are the ones in which mortality occurs and it is those cases that cause so much trouble and discussion. The study and discussion of appendicitis has caused a marked reduction for the past three years in the mortality due to appendicitis and its complications. DeCourcy, in 1936, in reporting 757 cases of all types operated upon during 1935, gave a reduction in mortality of 1.1 per cent from that of 1931, and for the perforated group a reduction of 5.7 per cent. "Walker found a mortality of 7.8 per cent in a review of more than 16,000 cases of appendicitis collected from the literature during the years 1907 to 1910, and a mortality of 5.3 per cent in 33,000 cases from 1927 to 1930." In Finney's series of 3,913 the mortality was 2.33 per cent. Garlock, in reporting 443 cases of acute appendicitis with a mortality of 4.6 per cent, called attention to the fact that in an earlier series of 755 cases the mortality was 6.2 per cent, a reduction of 1.6 per cent in

the later series. The cases of the two series were practically of the same type. He attributed the reduction in mortality to more carefully supervised and actively extended postoperative care of the sick cases. The average mortality in the 2,921 cases reviewed and reported by Reid was 9.53 per cent for the years 1915 to 1921 inclusive and 6.3 per cent for 1922 to 1933 inclusive. The average mortality for the last 12 years was 5.4 per cent. Pool, in reviewing 757 cases operated upon at the Employees' Hospital from November, 1919, to September 1, 1935, said 500 cases of the series were operated upon up to 1930 with a mortality of 7 per cent and 257 cases from 1930 to September 1, 1935, with a mortality of 3.9 per cent. In 1,133 operative cases of appendicitis reported by Haggard, the mortality was 2.5 per cent. In a later series of 2,211 cases up to September 1, 1934, the mortality was 3.39 per cent. Gile and Bowler, in reporting 791 operated cases with a total mortality of 1.89 per cent, called attention to the high mortality, 36.4 per cent, in 22 cases of perforated appendicitis with general peritonitis, and a mortality of 6.45 per cent in 93 cases of gangrenous appendicitis with local peritonitis with or without perforation. Coller and Potter reported 336 cases in which there were 14 deaths. There were 85 peritonitis cases that were designated as the deferred operation group with a mortality of 9.3 per cent.

Intestinal Decompression Through the Natural Channels.—In a short paper on spreading or general peritonitis of appendiceal origin read before the Jefferson County Medical Society in 1917, the writer reported 15 cases with one death. Six of the cases followed intra-abdominal rupture of appendiceal abscesses. They were all operated upon and all recovered. The following case represents fairly accurately this group of the series:

Case 15.—Elvin H., white, male, age 11. Admitted, Hillman Hospital, October 25, 1917. An older brother had died of a similar attack six months before following an operation for it. Present illness began with a chill, pain in abdomen, nausea and vomiting weeks before admission to hospital. On admission: Temperature 99 3/5°F. Pulse 102. Respiration 28. Urine negative. Leukocyte count 8,600. Abdomen greatly distended, rigid and very painful on palpation. Diagnosis: General peritonitis. Patient was immediately operated upon. Operation.—Right rectus incision. A large quantity of thick pus, which seemed to fill the entire abdomen, escaped. A ruptured necrotic mass was well up under the liver. This represented a ruptured retrocecal appendiceal abscess. A small stump of the ruptured gangrenous appendix was in the center of this mass and large fecal concretions or stones were in the mass. The stump of the appendix was ligated and amputated and buried with a catgut purse string suture. Free drainage was provided. The boy was nervous and emaciated when he underwent the operation. He had attacks of convulsions and his recovery was slow, but he finally recovered. He was well and went home six weeks after the operation.

Nine of the cases had a spreading or general peritonitis due to ruptured appendix. All recovered except one. The following case is representative of this group of the series of patients:

Case Report.—Paul P., Negro, male, age 13. Admitted, Hillman Hospital, April 27, 1915. Complaint: Generalized pain in abdomen. Present illness began with a chill seven days before admission. Patient was nauseated and vomited at intervals. Had high

temperature and pain in abdomen. Bowels had moved once or twice daily. On admission: Temperature 102 1/5°F. Respiration 54. Pulse 144. Abdomen greatly distended and tympanitic, no marked tenderness on pressure. Urine negative, leukocyte count 12,000, heart and lungs negative. Diagnosis: General peritonitis. Patient was operated upon immediately. Operation.—On opening the abdomen, a copious quantity of pus escaped. A large necrotic ruptured appendix was removed and free drainage provided for. Patient was well and went home four weeks after operation.

In operating upon these cases, light ether or gas oxygen anesthesia was used and usually the McBurney incision. Free drainage was provided by the use of light cigarette and soft fenestrated rubber tubes, one of each in the pelvis and the kidney pouch. The patient was placed in the Fowler position and on the right side, extreme semiprone position, the left knee flexed. The back supported by a pillow. This semiprone, right side position was maintained constantly 24 hours when the patient could be on the right side or turned slightly to the left at intervals but not on the back or left side under any circumstances. The dressings were changed frequently. The stomach was washed out, if indicated, before or after the anesthetic was given. Hypodermoclysis of normal salt solution or intravenous normal salt was given as indicated. The intestinal distention was relieved by starting the Murphy drip immediately after the operation, using a large size male catheter. Every 30 minutes or hour, or at frequent intervals, the rubber tube, with a length sufficient to reach the drainage bottle or receptacle on the floor attached to the glass nozzle connected to the normal salt container, was detached and lowered to the drainage receptacle, and in this manner, the gas and intestinal contents were removed by siphonage. It was found that intestinal drainage would be established almost immediately. The Murphy drip and the siphonage may be carried out by the use of the indicated rubber tubes attached to a Y tube; the clamps attached to the rubber tube connected with the normal salt or irrigating container and the rubber tube leading to the drainage receptacle being alternately tightened or loosened. The distention would become gradually less and the part of the abdomen distal to the drain would within a short time, six to 24 hours, become soft and comparatively painless. The writer's idea is that by this method of intestinal decompression through the natural channels, the toxins are removed from the intestinal tract and the intraluminal and intramural pressure is relieved and the necessary fluids are supplied. It is found that by this method it is rarely necessary to wash out the stomach. The drainage tract becomes a walled off open abscess. The cigarette drain may be removed or removed gradually after three to five days and the rubber tube can be removed gradually. If the toxins are being absorbed from the drainage tract as indicated by subnormal or elevated temperature, it is irrigated once or twice per day using a Luer syringe with a male catheter attached, which can extend to the bottom of the drainage tract. The drainage tube provides for the outflow of the normal salt or other irrigating solution. Aseptic and antiseptic technic should be scrupulously observed.

The writer has used the above method of treatment in more than 1,000 operated and non-operated cases of peritonitis of pelvic origin with less than 1 per cent mortality and in a large number of cases of peritonitis of appendiceal origin with a low mortality. The following cases are typical cases of spreading or general peritonitis:

Case Report.—Mr. A. C. B., South Highland Hospital. April 14, 1925. In consultation with Dr. J. D. Heacock, I saw this patient at 3:30 P.M. The night before, he was awakened with severe epigastric pain, nausea and vomiting, and the following day about noon, he had a very tender mass in the right iliac region. The symptoms had not abated up to the time Doctor Heacock asked me to see the patient. However, when I examined the patient, he was perfectly comfortable and smoking a cigarette. He said he was not sick, that he was entirely relieved and he was going to get up and go to town. I agreed with Doctor Heacock in his diagnosis and I told the patient that his appendix had ruptured and that he should be operated upon immediately. He declined. Doctor Heacock was advised of my conclusions and I made the suggestion that the patient be given absolutely nothing to relieve any pain which, I was sure, would develop shortly. At 3 A.M. the following morning (about 12 hours after his appendix had ruptured), I was told that the patient was in the South Highland Infirmary and seemed to be in the greatest agony. His abdominal muscles were intensely rigid. There was cold perspiration and he thought he surely was going to die. I advised immediate operation. His wife insisted that I wait until her mother could arrive at 9:30 the next morning. This I declined to do. I operated upon him under gas-oxygen anesthesia through a right rectus incision, and a copious quantity of pus exuded and was aspirated from the abdominal cavity. Soft rubber tubes and cigarette drains were placed in the pelvis and in the kidney region next to the abdominal wall and covered by omentum. The treatment was carried out as outlined in such cases. He was well and went home in three weeks. However, he developed a hernia at the site of drainage for which I successfully operated three months later.

Case No. 37406.—South Highland Infirmary, Miss M. B. B., age 25. Admitted November 13, 1925. Diagnosis on admission: Ruptured appendix with spreading or general peritonitis. Present attack began November 11 with cramping pain in abdomen which became localized over McBurney's point with nausea and vomiting. Had for a number of years recurring attacks of indigestion and of abdominal pain. On admission: Temperature 101.8°F., pulse 100, respiration 20. Physical examination was negative except distention and rigidity over entire abdomen; tenderness was greatest over right lower quadrant. Blood pressure 135/60. Leukocytes 13,750, lymphocytes 2 per cent, neutrophils 96 per cent, transitional 2 per cent, urine negative. Operation.—November 13, ether anesthesia, McBurney incision, large quantity of free pus in abdomen, which was aspirated. The ruptured appendix was removed, stump invaginated with purse string, two cigarette drains were placed—one in the right pelvis, one in the kidney region, two pieces of gauze in either angle. Pathologic report: Appendix ruptured and showed dark brown fecalith 1.3 cm. in its greatest measurement. The fecalith was laminated and calcareous. Diagnosis: Gangrenous ruptured appendix with fecalith. Patient discharged December 22, 1925, with slight drainage from incision but practically well.

Case No. C-4108.—St. Vincent Hospital, Mr. H. L. T., age 44, physical director, Birmingham City Schools. Admitted September 19, 1926; discharged October 10, 1926. Diagnosis on admission: Ruptured appendix with spreading or general peritonitis. Diagnosis on discharge: Ruptured gangrenous appendix with extensive area of gangrenous cecum, and spreading or general peritonitis. Present attack: Began Wednesday night, four days before admission, with sudden pain all over the abdomen. Patient had been to a picture show and on returning home had a large watery bowel move-

ment. He had not taken any purgative, but the next morning he took a large dose of Epsom salts and a bottle of citrate of magnesia. The pain became somewhat relieved in 36 to 48 hours. Friday morning, the second morning after the night of the initial attack, at 11 P.M., he had a chill followed by fever. He continued having fever and pain in the abdomen. He had similar attacks in January and April, 1926. On admission: Temperature 101°F., pulse 84, respiration 26, abdomen distended and exquisitely tender. Laboratory findings: Leukocytes 10,200, lymphocytes 13 per cent, large mononuclears 3 per cent, neutrophils 84 per cent, urine negative. Operation: Patient was operated upon one hour after admission: Gas-oxygen and ether anesthesia, McBurney incision, muscles not cut but widely separated, long gangrenous appendix with multiple ruptures removed. Appendix base and large area of cecum invaginated with two layers catgut sutures. Two cigarette and two tube drains placed—one in pelvis and one in kidney pouch. Gross findings: Ruptured gangrenous appendix with extensive area of gangrenous cecum. Free foul smelling pus in abdominal cavity. Patient was well and went home in 21 days.

Not infrequently patients are sent in in whom a diagnosis has been made of acute appendicitis and yet the symptoms are so atypical that the diagnosis is uncertain until the patient is thoroughly studied. This is especially true in pelvic appendicitis. Patients with pelvic inflammatory diseases also have an appendix and some of these cases may have acute appendicitis which finally ruptures, producing general or spreading peritonitis in addition to the peritonitis of pelvic origin. The writer has operated upon a number of such cases with acute appendicitis, ruptured and upruptured, limiting the operation to the appendix and drainage. Cases of pyelitis or urethral stricture may be referred with a diagnosis of appendicitis, and all the symptoms may be due to the urologic condition or the urologic condition may be present and the acute appendicitis also. Such patients should be carefully studied and be operated upon when, and if, indicated. The writer has had two cases of acute appendicitis in women with pulmonary tuberculosis, in each of whom pyelitis was also present. One case had a purulent pelvic appendix and the other had all the symptoms of acute appendicitis with pyelitis, and after consultation with a urologist and an able surgeon, it was decided that there was surely an acute appendicitis which should be operated upon without delay. The appendix, when removed, was designated by the pathologist as chronic. The patient, however, improved rapidly after the operation, whereas she had grown progressively worse previously.

In all the series of cases studied by the writer, it is clearly evident that peritonitis due to appendicitis, be it local, diffuse, spreading or general, is the real cause of the marked mortality due to appendicitis. To prevent peritonitis of appendiceal origin is the real appendix problem, and to do this there must be an early diagnosis and an appendicectomy before the infection extends beyond the appendix wall. The cases of appendicitis with complications—diffuse, spreading and general peritonitis and appendiceal abscesses—are the ones that test the judgment and skill of the greatest surgeons and they (the surgeons), the patients and the family of the patients, have, do and will continue to recognize the saving of lives in such cases, as entitling surgery to

the designation "The Queen of the Arts" and the surgeons who render the service as masters of the science and art of surgery.

The cases reported in the following series are all operative cases. The operations were performed by the members of the staff of each hospital. All cases were private except those in the Hillman.

HILLMAN HOSPITAL APPENDICITIS CASES 1913–1925 (Inclusive)

		Per Cent
Number of cases	1,664	
Number admitted with incorrect diagnoses. No operation	260	16.67%

	Number of Cases	Per Cent of Total Cases	Number of Deaths	
Appendicitis, acute (with abdominal complications of appendiceal origin, all of which were drained)	616	43.87	91	14.7%
Appendicitis, acute (clean—not drained)	788	56.12	1	0.0012%
Total operated cases	1,404		92	5.73%

COMMENT.—(1) It was thought that the number of cases for the period would be much larger but this was due to the length of time the complicated cases remained in the hospital (3 to 6 weeks), and the nursing and professional care given them.

(2) The high mortality in complicated cases.

(3) The low mortality in the clean undrained cases.

(4) The large number of incorrectly diagnosed cases admitted to the hospital.

(5) The clean case that died was a preoperatively undiagnosed case of Endameba histolytica. Endamebae were found in the appendix wall, and on autopsy multiple cecal ulcers had ruptured and there were multiple amebic abscesses in the liver.

HILLMAN HOSPITAL APPENDICITIS CASES January 1, 1931–October 1, 1936

Number of Cases	Type	Non-drained	Abscess	Local Peritonitis	General Peritonitis	Deaths	Year
233	Acute	224	13	0	12	8	1931
41	Chronic	40	0	0	1	1	1931
274		264	13	0	13	9	Total for 1931
301	Acute	298	27	0	6	12	1932
83	Chronic	80	2	0	1	1	1932
384		378	29	0	7	13	Total for 1932
276	Acute	269	30	0	8	4	1933
41	Chronic	0	3	0	0	2	1933
317		269	33	0	8	6	Total for 1933

HILLMAN HOSPITAL APPENDICITIS CASES—*Continued*

Number of Cases	Type	Non-drained	Abscess	Local Peritonitis	General Peritonitis	Deaths	Year
272	Acute	260	23	6	12	11	1934
96	Chronic	95	0	0	0	1	1934
368		355	23	6	12	12	Total for 1934
275	Acute	251	37	15	17	24	1935
79	Chronic	78	1	0	0	1	1935
354		329	38	15	17	25*	Total for 1935
222	Acute	208	11	5	11	17	1936
33	Chronic	32	0	0	0	0	1936
255		240	11	5	11	17	Total for 1936, up to October 1.
1,952		1,835	147	26	68	82	Grand total for 6 years

Mortality 4.2 per cent

* Causes of death for the year 1935

General Peritonitis 13—52%

Local Peritonitis 4—16%

Abscess 6—24%

Appendicitis with myocardial failure and pneumonia 2—8%
(1 acute appendicitis with myocardial failure. 1 chronic with pneumonia)

WEST END BAPTIST HOSPITAL APPENDICITIS CASES 1931–1935 (Inclusive)

Total number of cases 778.

Type of Case	1931	1932	1933	1934	1935	Total Cases
Chronic and subacute	57	30	26	35	45	193
Acute	119	94	75	81	96	465
Ruptured	5	2	2	5	8	22
Gangrenous	21	23	18	13	23	98
Deaths*	3	2	2	3	2	12

* All deaths were in ruptured gangrenous or supprative cases.

REPORT OF GORGAS BRANCH FOR FIVE YEARS

Total number of cases 696. Deaths* 3.

* Two deaths were in the gangrenous supprative groups and one death in the chronic group.

NORWOOD HOSPITAL APPENDICITIS CASES 1920–1937

Series 1920–1933	Number	Per-centage	Days Ill before Admission	Hospital Days	Deaths Number	Per Cent	Cause of Death
Acute	1,725	67.3	....	....	20	1.15%	
Subacute	243	9.6	....	....	0	0	
Ruptured	220	8.3	....	....	19	8.63%	
Chronic	354	14.8	....	....	0	0	
Total	2,542	100.0			39	1.10%	
Series 1933–1937							
Acute	221	29.9	18.7	8.4	3	1.35%	2 Penumonia 1 under anesthetic

NORWOOD HOSPITAL APPENDICITIS CASES—*Continued*

	Number	Per-centage	Days Ill before Admission	Hospital Days	Deaths Number	Per Cent	Cause of Death
Gangrenous.	140	18.8	36.2	10.3	1	0.71%	1 under anesthetic
Ruptured...	25	3.5	56	18.5	2	8.00%	Peritonitis
Abscess.....	20	2.5	120	15.3	0		
Subacute...	141	19.0	34.6	7.8	0		
Chronic.....	69	9.4	105	8.4	0		
Removed at other operations	126	16.9	....	....	1	0.80%	Undetermined
Total...	743	100.0		7	7	0.94%	
1920–1937							
Total.....	3,285	.....	....	....	46	1.40%	

SOUTH HIGHLANDS INFIRMARY APPENDICITIS CASES 1934–1936

	Number	Number with Peritonitis	Deaths	Per Cent
Appendicitis, gangrenous and perforated.....	117	2	4	3.4%
Appendicitis, with abscess.....	20	1	4	20.0%
Appendicitis, acute.....	451	1	3	0.66%
Appendicitis, chronic (recurrent)...	473	0	2	0.4%
Total.....	1,061	4	13	1.2%

ST. VINCENT'S HOSPITAL APPENDICITIS CASES 1932–1936

	Number	Number with Peritonitis	Deaths	Per Cent
Appendicitis, gangrenous and perforated.....	180	2	2	1.1%
Appendicitis, with abscess.....	58	0	1	1.7%
Appendicitis, acute (1 of these patients had a pulmonary embolus).	641	1	2	0.32%
Appendicitis, chronic.....	576	0	2	0.5%
Total.....	1,455	3	7	0.5%

CONCLUSIONS

(1) Intensive study by surgeons is being given to appendicitis and the mortality due to appendicitis is being markedly reduced as shown by the records of official statisticians and by the reports of large numbers of cases from many general and private hospitals and by individual operators.

(2) The mortality in the acute uncomplicated cases of appendicitis where the infection is limited to the appendiceal wall is very small, less than one half of one per cent.

(3) Cases of appendicitis with complications are the ones in which a mortality is likely to occur. More than 90 per cent of the mortality in most series of cases reported is due to some form of peritonitis.

(4) Some surgeons report as good results with the immediate operation after the indicated preparation of the patient as other surgeons do by the deferred operation with the indicated treatment. It is possible that the lack of agreement among surgeons relative to the two procedures—the immediate operation after the indicated preparation and the deferred operation with the indicated treatment—is more apparent than real. It is generally recognized now by surgeons that every patient should be properly prepared before an operation is performed, and this preoperative preparation and treatment may be what some regard as the deferred operative procedure.

(5) A broad, comprehensive, continuous, orderly, ethical, educational campaign, with reference to appendicitis as a widely prevalent disease, should be conducted by the national, state, county and city health authorities and by the schools, colleges, universities and industries throughout the country.

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DISCUSSION OF PAPERS OF DOCTORS MCCLURE, BUNCH, DOUGHTY AND HOGAN

DR. HUBERT A. ROYSTER (Raleigh, N. C.).—These papers are very interesting, and yet they all begin at the wrong end. I have said repeatedly that I would take my chances, and so would you, with a mediocre surgeon if I had an unruptured appendix, rather than have the most expert surgeon at the eleventh hour. The real truth is that none of us pays much attention to the pathology of the disease. The fact is that every acute appendicitis attack that comes to the operating table has been preceded by a pathologic condition going on for at least two years. When we question the patient: "Is this the first attack you have ever had?" Ninety per cent of them will say, "Yes," but when he says "first attack" he means the "worst" attack. Appendicitis is a going concern, a continuous process, and the acute attack is not the beginning, but the culmination of the disease. It represents the knock on the door saying, "Let me out." We have become convinced that the mild attacks, so called, do as much damage as the acute attack. It has been shown by Rogers of the New York Life that only 2 per cent of those who have one attack escape others. Once appendicitis always appendicitis, until the little criminal is executed.

It is not up to us to say that these cases cannot be diagnosed; they can, for suspicion is better than cardinal symptoms. When Doctor McClure speaks of 66 per cent of atypical cases, I reply there are no atypical cases. We must go behind the returns. All our educational work is part of this problem. When the patient has the terminal type we have come to the end of the pathology instead of the beginning, because acute appendicitis is, as Dieulafoy said in 1898, an infection of a vestigial organ structure, a pathologic process in a closed tube, and there is no escape for it. Once damaged it is always damaged. So, when we speak of the different forms of appendicitis, even atypical appendicitis, we can say with Hendon, "the only hand that paints a perfect pathologic picture is the hand of death."

DR. JOHN T. MOORE (Houston, Tex.).—I have learned a good lesson from these three excellent papers. I do not know whether any of you will remember our colleague, Dr. R. T. Morris of New York. I have just read with great pleasure "Fifty Years a Surgeon." This book causes me to go back over a lot of things I remembered when Dr. A. J. Ochsner and others were discussing appendicitis. Everything practically seems to have been said about the pathology, the symptoms and so forth, but it all comes back to a few things which I have tried to instill into the minds of the coming surgeons in our State. The first of these is to avoid purgatives before the diagnosis is made. The other one is that after making the diagnosis, there should be an immediate operation, provided you know how to operate properly. Where we run into a discussion, in our country, is in regard to the kind of incision that should be made in acute appendicitis. If these studies show anything in the world it is what Morris called attention to when McBurney and other great surgeons were having a death rate of 10 to 20 per cent, when Morris had a death rate of less than 1 per cent. He used a button hole incision and a finger in the abdomen. We have tried to teach our students that the incision for acute appendicitis is the small McBurney incision. Get the appendix out and never let the ileum come in contact with anything septic. If these studies show anything, it is that 10 to 20 per cent of the cases operated upon were operated after the third day, and then I would be interested in knowing what the incision was. It has been customary with us to operate upon those cases as soon as we see them, but we try not to see anything but the cecum.

You can make the McBurney incision far out, and in the majority of cases can get the appendix out without coming in contact with anything but the appendix.

The question of drainage has been studied by members of this Association. Some drain and some do not. It has been our custom in the infected cases where we see serum that seems to contain pus, or in the gangrenous appendix, to put in a drain.

We want to teach our younger men that the right rectus incision is an exploratory incision and not an incision for taking out an appendix. If we can get that over, our mortality will be lower. The reason our mortality ran up so high in the past few years was because we stopped emphasizing the particular things that Doctor Finney and some of the others have emphasized many years ago—when to operate and how to operate. Since you have started this discussion again, it is very pleasing to know that the statistics in Birmingham are so very good. I would be interested in knowing how many of these cases are operated upon by highly trained surgeons and how many by general practitioners. In our part of the country our mortality is high because there are so many operations by general practitioners. That must be emphasized to make the mortality lower.

DR. ROBERT L. SANDERS (Memphis, Tenn.).—I wish to emphasize what Doctor Moore has just said regarding the McBurney incision. Such a procedure is not only time saving, less disabling from the standpoint of postoperative herniae, *etc.*, but it is actually life saving in many instances. I am convinced that we have a lower mortality rate in the cases of acute perforated appendicitis when they are approached through such an opening in the abdominal wall.

I go a step farther and leave the gridiron wounds completely open in all badly infected cases. When the appendix is perforated and free pus is in the abdominal cavity, the appendix may or may not be removed according to the indications. Penrose drains are placed in the wound, the peritoneum loosely closed about them to prevent the escape of intestinal loops, and the muscles, fascia and skin are left open—no sutures being used. Pus follows lines of least resistance and comes to the surface unobstructed. Such wounds soon clean up, no fascia is lost, and healing is usually prompt and complete—herniae are rare—secondary closures are seldom necessary. To hasten the recovery, wounds may be drawn together by adhesive strips after the infection has subsided. In contrast to this procedure, one may close the muscles, fascia and skin about the drains and observe pus burrowing beneath the fascial planes, infecting and devitalizing all structures. About the fifth day, a large swollen area may be present, and when the sutures are removed, foul infected fluid will escape, and dark fascia may be seen for a distance of several inches. For days, and even weeks, strips of dead fascia may be removed, leaving a gap that is not easily bridged; potential, if not actual, herniae may result. Doctor Bunch mentioned loose closure of the wounds. In our cases we have found it much better to refrain from using sutures at all, for the above reasons. Some years ago, Doctor Bancroft read a paper in which he advocated nonclosure of such infected wounds. He studied the incidence of postoperative herniae and found them much less when no sutures were used in the abdominal wall. Our mortality has been materially reduced since we adopted this method of treatment.

One other point in regard to Doctor McClure's paper. I thoroughly agree with him in the use of pitressin in cases of acute perforated appendicitis

with peritonitis. Intestinal muscle tone is kept up, ileus is not so marked, and distention is prevented in almost all cases in which it is used. We give one cubic centimeter subcutaneously when the anesthetic is started, repeat it in two hours after the patient has been returned to bed, and then every four hours until six doses are given. We have been much pleased with the effect.

DR. R. S. HILL (Montgomery, Ala.).—I feel safe in saying the progress of American surgery has been satisfactory and gratifying in every respect except in the reduction of the mortality from appendicitis. This is particularly regrettable in that there is no other surgical condition of greater practical importance. Through the many years since the epoch making article by Reginald Fitz (1886), fixing the minds of the profession on appendicitis, the "what," the "how" and the "when" to do have been discussed without a satisfying result in reducing the mortality from the disease. It is not my purpose to discuss the "what" and the "how" to do, but the "when" to do—to operate. I am convinced that the death rate from appendicitis will be materially lessened if these patients are more promptly brought to operation.

Let it be gotten over to the nonsurgical medical men that appendicitis is a distinctly surgical disease which, in the absence of some definite individual contraindication to operation, calls for prompt surgical intervention and that efforts to avoid operation are unwarranted and dangerous. Furthermore, in the exceptional case, in which a positive diagnosis cannot be readily made, the surgeon should have the moral courage to operate on a probable diagnosis rather than subject the patient to the great danger of suppuration from delay in an effort at refinement in diagnosis.

To drive home the thought I have, permit me to draw from material furnished by Doctor Finney in the report—in his own inimitable way—of two patients to this Association a few years ago. Having been called out of the city to perform an appendicectomy, he said, after the operation, to the doctor in charge: "Doctor, if and when you find out what is the matter with this child will you wire me at my expense?" The next day he received a one word telegram: "Measles." With this case fresh in mind, he was called to operate upon another child. He asked if the patient had been exposed to measles. He had been, and in fact, "There is a case in the house now." "Well," said Doctor Finney, "we will wait developments." A pus appendix was the result. Now if, on the theory, "it is human to err," I may have the temerity to use the word error in connection with Doctor Finney's surgical work, I would say he did not make a surgical error in the first case, for his operation added little to the danger and prevented what possibly might have been a serious risk as exemplified in the outcome in the second patient.

Many years ago, Doctor McBurney operated upon the wife of a friend of mine for gallstones and found no trouble with the gallbladder. It was one of those cases discussed by Dr. Maurice Richardson, who, by the way, seemingly preferred talking of his mistakes rather than his successes, in a paper entitled "Operating for Gallstones When There Are No Gallstones." The husband objected to paying Doctor McBurney's bill, as no gallstones were found. Doctor McBurney replied, "Why, my good fellow, it is worth that much to know your wife has no gallstones—with this trouble eliminated we can better discover and treat the condition causing your wife's ill health."

In what I have said, do not understand me as advocating the reckless removal of the appendix or of underestimating the desirability of an accurate diagnosis. I am simply advocating prompt operation—giving the patient the best chance to recover, even though there is a possibility of an occasional unnecessary appendicectomy being performed.

DR. ROY D. MCCLURE (closing).—Our hat is off to North and South Carolina for, as the first table indicated, they have the lowest mortality rate in the United States from appendicitis.

I was interested in Doctor Royster's remarks, that we in our discussion do not go back far enough. In the past nine years through the work of our assistants in the Medical Division at the Ford Rubber Plantation in Brazil, we have had the opportunity to study the statistics on several thousand natives living and working in primitive conditions in the jungle.

Not a single case of appendicitis developed during the first year, but each succeeding year has shown a definite increase in the incidence of acute appendicitis, and at the present time cases of gangrenous appendicitis are not uncommon. The living conditions of these employees have steadily improved according to our standards of living. The most noticeable change has been in the food habits and this has been the substitution of fresh meat and canned goods for the usual native diet of fruit and fish.

Some years ago I remember the tendency to scoff a little at statistical studies and it was not infrequently stated that "statistics could be made to prove anything." However, I feel sure that the ultimate decision of the surgical world as when to operate on the patient with a perforated appendicitis and peritonitis can only be made by the honest and accurate comparison of statistical studies. At this meeting, in which each of us set forth our results for your study and consideration and the exchange of ideas and experiences in this discussion, are steps toward reaching that ultimate decision. The expectant or Ochsner method of treatment might be better in some of the older group of patients where abscess formation is more apt to occur. Again it might not be so good in children where an abscess is not so apt to occur, as is shown in our series of cases.

Perhaps when the American Board of Surgery for the certification of surgeons being set up by the Southern Surgical in coöperation with other leading surgical societies is instituted, the mortality rate will decrease. At least that is the worthy aim and hope of this association and the other surgical societies. This could not come in our generation but we can set up certain standards in surgical training and practice which may very well bear fruit in the next generation in which our children and grandchildren must live.

EPIGASTRIC SYMPTOMS IN ACUTE LUNG AND HEART DISEASES

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RECENT medical literature shows that there is a renewed and active interest in the general subject of acute pain in the upper abdomen. The newer conception of cardiac pathology in reference to angina pectoris and coronary disease has been reflected in widespread study of the meaning and interpretation of severe epigastric pain. Although Heberden, in 1768, wrote a very clear and concise description of coronary sclerosis, and before him Jenner won a wager with another physician that his patient died from the disease, the pathology of coronary disease was forgotten or overlooked until about 1912, when adequate descriptions and case reports began to appear in German medical literature. Herrick,[2] in 1912, wrote the first report giving convincing evidence. Prior to this, Dock, in 1896, recognized a case and proved it by autopsy. Osler,[3] in 1910, understood the pathology of coronary thrombosis and described accurately the significance of the associated epigastric pain.

A brief review of the text-book literature of this period shows that the relation between cardiac disease and upper abdominal pain was not understood nor described. Hirschfelder,[1] in the third edition of his book published in 1918, Mackenzie,[4] in 1917, and others of equal eminence do not mention the subject. Among the surgeons may be mentioned Walton,[5] who does not speak of the heart when describing upper abdominal perforations; Cope,[6] as late as 1935, omits any mention of the heart when writing of the diagnosis of the acute abdomen. However, Vaquez,[7] in 1924, a medical writer, gives a very comprehensive and complete description of the pathology and the symptomatology of the accepted views of the heart diseases which exhibit symptoms in the upper abdomen. Cecil,[8] in 1934, gives the symptoms accurately but does not emphasize the importance of the symptoms in differentiating between a medical and a surgical case. LeCount,[9] in 1918, gives a study of the pathology which is very complete and lays down the basis for the symptomatology of coronary sclerosis and thrombosis. It has long been known that aortic disease, under some conditions, produces severe epigastric pain which may resemble in every detail the pain of coronary obstruction. This is also true of aortic aneurysm, disease of the cervical spine,[10] and Salzmann[11] reports from cases of ruptured heart and cases of ruptured aneurysm which presented epigastric symptoms suggestive of a perforated viscus.

Embryology and Anatomy.—In the second week,[12] the embryonic cell mass divides into three systems, *i.e.*, entodermal, ectodermal and mesodermal, from which the structures of the body develop. In the third week, the celomic

cavity forms, which is the primitive representation of the pleural, peritoneal and the pericardial cavities in the adult. This is produced by division into two layers of the mesoderm, the inner of which in association with the layer of entoderm forms the *splanchnopleure* and an outer wall in association with a layer of ectoderm forms the *somatopleure*; these membranes enclose the abdominal and thoracic cavities.

In the fifth week, the heart and lungs have been formed and occupy a position in the neck, the lungs above the heart. They lie beneath the chin and the larynx; during the next three weeks, the heart passes from a subpharyngeal position to the adult position in the chest. The lungs follow the heart downwards but remain above it. While this is taking place, the neck elongates and the heart lies about the middorsal region of the spine with the lungs on either side of it. All of the connecting structures in the chest become elongated to conform to the heart and lungs, *i.e.*, the esophagus, the trachea, vagus nerves, jugular veins and carotid arteries.

The embryology of the lungs is more complicated than that of the heart: in the earliest stages of development, the lungs are represented by the branchial system which has to be completely changed into an air breathing system. The changes are going at the same time as the development of the heart and are becoming cooperative. The lungs arise from the analogue of the swim bladder in fishes and during the fifth week it is bifid, each half being properly called a lung. They lie in the abdominal cavity above the heart and the liver; a respiratory passage develops, leading from the pharynx to the lungs, and a vascular system develops, an artery to each lung, from the sixth visceral arch. The branchial muscles which formerly forced water through the gill slits now become pharyngeal muscles and pump air into the lungs, acting as muscles of inspiration. The muscles of the body wall are formed into muscles of expiration. They are formed from the primitive muscle sheets, two of which are of interest, namely, the transversalis sheet which arises from the spine and ends in the pericardium, esophagus, and the roots of the lungs and the deep lamina of the rectus abdominis muscle which ends in the pericardium, the first of which becomes the diaphragm. The nerves supplying these muscles come down along the vena cava. At this stage, and following, the diaphragm develops and separates the chest viscera from the abdominal and the diaphragm becomes an organ of inspiration. A striking factor is that as the heart and lungs descend, they carry with them their original nerve supply and a modified vascular supply. In the sixth and seventh weeks, there is rapid expansion of the lungs downwards with the development of the celomic cavity.

The nervous system, the parasympathetic, which supplies the heart and lungs comes from the basal centers and descends with the organs. The symptoms, arising from disease of these viscera, radiate over regions of the body that are not supplied by the basal nerves but these have connections with the sympathetic nerves and their ganglia which explain their distribution. This explains the segmental character of the symptoms which are located in the body wall, in the epigastrium and in other regions of the body.

The Nerves Directly Concerned with Cardiac Function.—The brief description by Herrmann[13] is recommended for its clarity and correctness, *i.e.*, "The most recent work seems to indicate that the important efferent tracts from the deep cardiac and aortic plexuses lead through the sympathetic connections and chain from the fourth to the first thoracic ganglia and their respective posterior spinal roots.

"The intricate network of nerve fibers and ganglion cells is spread over various tissues about the great vessels and the heart. From the base of the aorta fibers are to be found spreading down along the coronary arteries and their branches just beneath the epicardium. The fibers and plexuses are of autonomic and sympathetic origin coming from the vagus, the recurrent laryngeal; the superior, middle and inferior cervical; the stellate ganglia, the inferior middle and superior cardiac nerves; the first to the fifth thoracic ganglia, their rami and the posterior spinal roots."

The nerve supply both to the heart and lungs is very intricate and confusing; but the clinical interest is centered in the distribution of pain and muscle disorganization in the coverings of the upper abdomen. The problem becomes very simple when stated in these terms. Nausea and vomiting, as well as suspected pain in the viscera, may be due to reflex action through the vagus, combined ganglia and the sympathetic nerves on the muscle walls of these organs.

The Clinical Distribution of the Pain Impulse.—The type and character of pain and its distribution to the epigastrium are the features of every case, first to attract the attention of the examiner. It is only when the pain during a supradiaphragmatic illness is reflected into the upper abdomen that the case becomes of surgical importance.

The vagus and the sympathetic nerves which supply the chest viscera are connected with abdominal musculature indirectly; the connection is central and through the ganglia of the sympathetics which connect with the segments of the spinal cord which excite the areas in the cord supplying the intercostal nerves that supply the musculature of the abdomen. Keith[12] says that undoubtedly the cord was segmented in very early evolutionary life. There is no question about functional segmentation which is expressed clinically in these cases under discussion. The essential nerve supply of the heart and lungs is from the basal centers which extend downwards as the vagus; the opposing system, the sympathetic, with its interminable connections between the ganglia, the many medullated and nonmedullated fibers connecting both systems, is distinctly segmented. The efferent nerve branches carrying impulses to and from the diseased areas reach the cord segmentally and the distribution of these painful impulses are through efferent fibers which transmit both pain and motor stimuli to the abdominal muscles. While it may be true that there are similar connections between the vagus and the sympathetic nervous systems supplying the epigastric viscera, the type of pain is not that which is seen in the abdomen during acute disease of these organs. In acute intestinal obstruction, acute appendicitis, acute pancreatitis and similar affec-

tions, *uncomplicated* disease of these organs is not attended by rigidity or marked tension of the abdominal muscles. Not until the parietal peritoneum has become affected does the musculature become rigid or even tense. Illustrative instances are impacted gallstones, the early stages of appendicitis, intestinal obstruction, *etc.*

During an attack of severe pain from any cause, there is always more or less general muscle tension all over the body; the patient holds his breath, clinches his hands, gazes intently, lies in a tense position, *etc.*, with greater tension in the region of the pain, or he is frantic and tossing, but always tense.

In acute inflammatory diseases of the abdomen, the area of peritoneum involved is always beneath the region of muscle tension; there is a definite connection between these occurrences; the pain is localized with tenderness and muscle tension. But, as long as there is no peritoneal irritation, as is so notable in the splanchnic stage of acute appendicitis, there is no muscle reaction. In other words, it requires peritoneal irritation to provoke muscle spasm and tension in the acute inflammatory diseases of the viscera. On the other hand, there can be moderate muscle tenderness, moderate rigidity and any degree of pain in the muscles of the upper abdomen which are supplied by nerves which are irritated either in the cord segments or along the fibers above the diaphragm. Since no one claims that the peritoneum lining the epigastrium or covering the viscera is irritated by the pathology above the diaphragm there could be no true muscle rigidity. The nerves concerned with the muscle tension and pain in the upper abdomen are the intercostals from the sixth, seventh, eighth, ninth, and tenth. The pain and tension are protective and defensive in their functions; since there is nothing to protect in the upper abdomen—the disease being above the diaphragm—the technic of examination as described elsewhere in this paper should differentiate between what is real and what is apparent.

Acute Inflammation of the Lungs and Pleura.—The patient is suffering from pain in the epigastrium which is of such gravity as to demand a differential diagnosis from some acute pulmonary ailment. There is usually nothing in the general symptoms to determine the reason for the epigastric findings; the temperature, pulse rate, appearance of the patient, type of breathing and the history are those of some acute infection.

The usual symptoms of pneumonia, such as high fever, pain in the affected side, rapid breathing, cough and limited diaphragmatic action, especially on the side involved, are noted. The significant and only symptoms of any importance are epigastric pain, muscle tension, and tenderness; these may be of any degree, from mild pain and tenderness to great severity; the muscle tension is never to the degree of rigidity. There is always the possibility that the patient is suffering simultaneously from pneumonia and some acute abdominal affection such as acute cholecystitis, acute appendicitis, acute pancreatitis, *etc.* The patient with pneumonia almost always gives a history of some pulmonary disturbance as the inaugural trouble.

The diagnostic differences between the muscle tension of lung affections

and peritonitis are that the muscle is never in marked or definite spasm. Gentle palpation will disclose some measure of relaxation between the respiratory movements, never in muscle rigidity; voluntary relaxation is possible with tension, never with rigidity; cutaneous tenderness and muscle tenderness may be identical in both conditions; deep tenderness, elicited by pressure with the flat hand increasing with attempted muscle relaxation, is absent in pneumonia; it is present in muscle tension from peritonitis. Percussion is of minor clinical significance as a differential diagnostic measure. The characteristic muscle fixation of the abdomen is not present in any lung affection, *viz.:* pneumonia, lung abscess, pulmonary infarct, pleurisy, *etc.* The abdominal signs and symptoms often met with in acute lung diseases are limited to the muscle wall and the problem in diagnosis is to bring out these features. I think that it can be made with great certainty. If the patient is suffering from two distinct diseases, pneumonia and appendicitis, for instance, the history of the onset and the chronology of pain will furnish all the required evidence for making the correct diagnosis in both instances as the symptoms are superimposed, but quite different. There is rarely, if ever, in acute infections of the lungs and pleura, or the sudden and tragic onset of epigastric pain as is the rule in cases of angina pectoris and coronary thrombosis.

Acute Cardiac Emergencies; Angina Pectoris and Coronary Thrombosis. These patients present quite a different clinical picture from the pneumonia patients. The onset of pain is usually sudden, very tense, located both in the left chest and in the epigastrium with radiations into the shoulders, arms, the neck, *etc.*, and is unrelieved by the ordinary doses of morphine or other drugs. There is often nausea, vomiting and respiratory distress. Here, as in the lung cases, the differential diagnosis is limited to the area of the upper abdomen. In some cases, the area of pain may be limited to the epigastrium and upper regions of the abdomen. The history may reveal a cardiac patient; but, as elsewhere, the patient may be suffering from two coincidental ailments, one of which is medical and the other surgical, and demanding immediate surgical attention. In this light, the diagnosis becomes of urgent importance.

In some instances of acute epigastric surgical emergencies, the symptoms may be reflected into the chest and confuse the examiner. But, in the painful cardiac diseases, they present such characteristic signs and symptoms that attention to the heart is mandatory. Among these are the absolute quiet of the angina pectoris patient in contrast to the restlessness of the coronary case; this, alone, should make the examiner alert because this condition of the patient is the characteristic attitude of the patient suffering from a perforated viscus. The patient with coronary thrombosis is restless and difficult to control in contrast to both the angina pectoris patients and those with a perforated viscus. There is often nothing characteristic in the heart examination of the angina pectoris case, but there may be evidences of a diseased heart in coronary thrombosis. The condition of the heart must, therefore, be carefully determined before a decision is reached. When the suspicion of the examiner is aroused as to the heart, the way is then made easy for the study of the abdom-

inal evidence. Shock may be of any degree in either cardiac or abdominal patients.

Here, too, as in the examination of the upper abdomen in acute diseases of the chest, the whole problem is simplified into the determination of muscle tone, tenderness, and pain. In perforation of the duodenum or stomach or the rupture of the gallbladder, the onset of symptoms is as sudden and as severe as coronary thrombosis or angina pectoris. This feature is characteristic of all three diseases. But, here, the similarity recedes and, with time, the symptoms pursue a different course. The suddenness of the pain of angina pectoris and coronary thrombosis is associated with terrifying sensations of impending death, pressure in the chest, dyspnea, radiation of the pain into the upper chest, into the arms and shoulders, or into the neck; whereas, while there may be moderate reflection of pain into the chest, there are not the same psychic and mental fears induced by the pain of a perforated viscus as in cardiac tragedies. There may be nausea and vomiting, abdominal distention, some degree of shock, muscle tension and tenderness with fever in coronary thrombosis, but the cardiac examination will, in the absence of definite muscle rigidity and expanding tension in the abdomen, clearly direct the examiner to the cause of the trouble.

With the gentlest palpation with the finger tips, and the constant pressure with the full, flat hand on the tense muscles, with the patient's cooperative attempt at muscle relaxation, there will be perceptible softening and relaxation of the muscle tone, the partial or even complete disappearance of pain and tenderness which is not the case when the epigastric symptoms are due to a ruptured viscus. There may be distention and the disappearance of liver dulness in both instances; however, this symptom has little value. A roentgenologic study will show escaped gas or fluid displacement if there is a perforation. Percussion and palpation, gently performed, are the best measures with which to diagnose the pain, muscle tension and skin and muscle tenderness whether the case is a perforated viscus or a cardiac case. The laboratory technic of whatever kind is of minor value in the presence of a suspected or an actual epigastric emergency whether medical or surgical. After the first few hours of diagnostic uncertainty, the progress of the disease usually becomes determinative one way or the other; it is in these stages that the diagnosis is so important.

The cases suffering from any lung disease have râles, rapid breathing, dulness in the lungs, limited breathing on the affected side; there is no muscle rigidity, no mass, no deep tenderness in the abdomen. There may be cutaneous hyperesthesia and hyperalgesia and marked muscle tension.

Differential Diagnosis.—The problem is among three main generic pathologic conditions, *viz.*: angina pectoris, coronary disease, and disease of the epigastric viscera. The first two are purely medical, the last one is purely surgical. The examiner is confronted with a definite emergency in any instance. There are other symptom complexes which may arise, but they are relatively unimportant.

It may be graphically represented in tabular form, which, however is not meant to be complete in every detail :[13, 14, 15, 16, 17, 18, 19, 20, 21, 22] (Table I).

TABLE I

DIFFERENTIAL DIAGNOSIS BETWEEN ANGINA PECTORIS, CORONARY AND EPIGASTRIC DISEASE

Angina Pectoris	Coronary Disease	Epigastric Disease
A perfectly still patient	Restless patient	Quiet patient
Symptoms of shock and collapse may be present	Shock is often present	Shock often present
Attacks come without effort	Without effort	Often after effort
No change in pulse rate, pressure rises	Fast pulse, low pressure	Little change in the early stages
Temperature normal	Fever may be present	Fever generally present, shortly after onset. Temperature may be subnormal
Gastric symptoms rarely present	Gastric symptoms often present	Nausea and vomiting the rule
Blood picture normal	Blood picture often abnormal	Blood always abnormal
Before 50 yrs. of age	After 50 yrs. of age	Any age after youth
Pain in chest, cardiac region, retrosternal, substernal, in arms, neck, elsewhere. Epigastrium in some cases	Pain comes suddenly, in same regions as in angina pectoris; epigastrium. Fine râles may be present in bases of the lungs. Never in right shoulder region	Pain comes suddenly, epigastrium first, diffuses downwards, may be in lower chest, often in right shoulder region. Never in arms, upper chest
Epigastric muscle tension, never rigidity, no mass	Muscle tension—not muscle rigidity; relieved with opiates. No mass. Liver dulness never absent	Epigastric muscle tension, unrelieved by opiates in ordinary doses; mass often palpable. Fluctuation often noted; liver dulness often absent
Hyperalgesia and hyperesthesia may be present	The same for coronary disease	Always present
Heart findings generally normal	Heart may show abnormal beats and sounds, friction rubs. Later, definite changes	Nothing significant
Pain relieved by vasodilators	Not relieved	No effect
Patient improves under treatment	May improve, or get worse	Patient gets worse
Abdomen generally mobile	Abdomen mobile; often some tension; no rigidity	Abdomen immobile; often board-like
Medical emergency	Medical emergency	Surgical emergency

The diagnosis of epigastric surgical injuries demands immediate decision; the life or death of the patient may depend upon what is done within a few hours after the onset of pain. Delay in making a diagnosis when the disease is above the diaphragm does not increase the risk or compromise the patient's welfare in any way.

CASE REPORTS

Case 1.—A male, age 30, was admitted to the hospital and gave a history of having been seized with a sudden, severe, excruciating pain in the upper abdomen associated with

nausea, vomiting, *etc.*, at six o'clock the previous evening. He was seen by a physician who gave morphine, something by the mouth and was told to take an enema. He suffered all night and came to the hospital about 9 A.M. the following morning. He was diagnosed as a case of coronary thrombosis. The patient rapidly got worse, consultation was called for and the diagnosis of a perforated viscus was made by the consultant. The advice was not followed until the patient got in extremis about 1 P.M., when the abdomen was opened and a very large perforation of a duodenal ulcer was found. The patient died.

Case 2.—A female, age 44, was taken suddenly ill with an excruciating pain in the epigastrium. The family physician gave her two half grain doses of morphine within two hours with very little relief. A surgeon was called to operate for a perforated epigastric viscus. The patient was very neurotic and had had four operations previously. The gallbladder had been removed for a similar attack. Subsequent studies showed that the heart gave the usual electrocardiographic findings of a small thrombosis of a branch of the coronary artery. Appropriate treatment relieved her condition.

Case 3.—The patient was seized with a sudden, severe epigastric pain, associated with the usual clinical findings of an epigastric lesion. He was 65, and had been a chronic arteriotic case. A surgeon was called to operate for a perforated viscus. The patient died within 24 hours, having evidenced a typical picture of a coronary thrombosis.

To these reports may be added several cases of right sided pneumonia, which were diagnosed acute appendicitis, one of which was operated upon for appendicitis; another was diagnosed acute cholecystitis, but the surgical consultant found a pleurisy with effusion. Similar cases could be added to these. Two of which were rather tragic as laparotomies were performed when the condition proved to be a painful heart. Acute surgical diseases in the abdomen may coexist with painful heart diseases.

Some Guideposts.—Every patient under 40 years of age presenting symptoms of an acute epigastric pathology should be examined for both lung and heart disease, but the chances are all in favor of acute disease of abdominal viscera; every patient over 50 presenting such symptoms should not be operated upon until the condition of the heart and lungs is ascertained.

It is noteworthy that males are more prone to the causative factors which produce coronary thrombosis and angina pectoris, and, also, that Africans, Asiatics and Mexicans are almost immune from such circulatory disturbances.

Jackson and Jackson[23] have advanced a theory of the causation of the pain in angina pectoris and coronary thrombosis. They report their experimental studies which appear to show that every symptom of pain, and its distribution, can be reproduced by electrical stimulation of the esophagus and, therefore, attribute all of the symptoms to esophageal lesions caused by stretching the muscles, lacerations and other injuries from vomiting, strains, spasms, *etc.* They attribute nothing to the pathology found in the heart or the great blood vessels.

SUMMARY.—(1) The basis of the distribution of the symptoms of pain and tenderness observed in coronary thrombosis and angina pectoris and allied lesions is the embryologic development of the human organism.

(2) When descent of the heart and lungs into the adult position takes place, the embryologic nerve supply is carried along with the organs and is not segmental.

(3) Later, but also early in embryologic life, there is developed a counteracting nervous system, the sympathetic, with its many ganglia and connecting fibers between the basal nerves and the sympathetic nerves, whose function is to correlate motor, sensory and functional impulses of these organs.

(4) Later, also, as the body elongates, there is developed a cerebrospinal segmented nervous system, which, with its numerous connections between the basal and sympathetic systems through connecting fibers, innervates the body wall and the serous lining, pleura and pericardium, whose function is, through its motor and sensory nerves, protection of the vital organs and to herald the presence of disease; these warning signs are pain, tenderness and muscular rigidity.

(5) There is an overlapping of these nerve fibers with consequent confusion of the symptoms largely because the basal nerves (parasympathetic) and the sympathetic nerves, through their ganglia, connect with the segmented cerebrospinal nerves which supply the body wall. There may be, also, true connecting fibers between these systems to the viscera which, under the stimulus of disease, may produce pain in the organs apart from the pain in the body wall; if true, such pain should be rhythmical and diffuse, which it is not, and separate from the sensations in the muscles.

(6) Muscle tension and rigidity due to irritation of underlying serous covering, or lining, is marked even to board-like rigidity and is definite and segmented and increased pressure heightens the pain and tension; whereas, irritation of serous membranes elsewhere, as the pericardium and pleura in the diseases under discussion, does not produce in the abdominal muscles extremes of muscle rigidity and tenderness and rarely, if ever, deep tenderness.

(7) A method of examination is described which should differentiate between muscle reaction and tenderness in cases of cardiac and pulmonary disease with symptoms referred to the epigastrium from acute surgical disease within the abdomen.

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REGIONAL ILEITIS

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WHEN Crohn[3] and his associates called attention to a granulomatous lesion of the terminal portion of the ileum, which they termed "regional ileitis," many of us recalled, from our own experience, isolated instances in which the features of the lesion conformed to this description. With greater care in history taking, improved roentgenologic technic, and surgical exploration, the number of cases of regional ileitis seen at The Mayo Clinic have rapidly increased. This may be partially accounted for by an increased incidence of the disease, but it is our conviction that greater alertness in recognizing the disease has been an important, if not the dominant, reason. It therefore seems that a review of our experience is worth while, even if it serves only to focus attention on this interesting problem.

There are two clinical types of regional enteritis: (1) The involvement of a rather short, localized segment, which usually consists of a single lesion, and (2) a similar process which involves longer segments and usually consists of multiple lesions. In addition to one or two short inflamed segments in the lower part of the ileum, we have included, in this category, involvement of similar segments which are situated in the upper part of the ileum and jejunum and which may be multiple or even involve many feet of the intestine (applying the term "regional enteritis"). Finally, the term may include involvement of one or more segments of the lower part of the ileum, which may spread to the cecum and ascending colon. The involvement may be either separate or continuous. Pathologically, both types differ grossly in extent, but microscopically both are associated with the same granulomatous process and tend to destroy all the intestinal walls, to cause stricture, and not uncommonly tend to cause adhesions to the adjacent bowel and produce fistulous formation.

Etiologically, there is as yet no final agreement. The first query always is: "Are you sure it is not tuberculosis?" To the best of our knowledge, this particular lesion is not tuberculosis. Repeated sections have been stained for the tubercle bacillus and in several of our cases, as well as those reported by others, guinea-pigs have been inoculated but there has not been any evidence of tuberculosis. If there is any doubt, we have made it a rule not to include such a case in our study. Felsen,[4] reporting his work with the Bacillus dysenteriae, expressed the opinion that this organism is

the cause. The correlation of cultures of the stools, agglutinating properties of the blood serum, and tests for the presence of bacteriophage in the stools offer several problems that make it difficult to be sure that the question is settled. Felsen's clinical description of cases in which the blood serum had positive agglutinating properties is certainly suggestive. A segment of bowel which was resected at operation was submitted for bacteriologic study in four of our cases. In one instance cultures from the thickened wall of the bowel yielded a pleomorphic gram-positive Streptococcus. In the other three instances cultures were negative. A similar finding was reported by Mixter,[5] who obtained an anaerobic Streptococcus from the peritoneal fluid, from a mesenteric lymph node, and from the deep surface of the intestinal ulceration.

Clinically, ulcerative colitis and regional enteritis are similar. In both, there is usually the history of early exacerbations and remissions. As time goes on, the disease becomes more continuous and more resistant to treatment. In both diseases the process may continue without remissions and one may encounter the small, short, narrowed colon (a small hose of 14 to 16 inches [35.4 to 40.6 cm.] in length), or may find six to ten feet (182 to 300 cm.) of greatly thickened, narrowed hose-like jejunum or ileum.

In our experience, we have seen both the acute and chronic stages of inflammation of the small bowel. We have records of several cases in which an exploratory laparotomy for acute appendicitis revealed a swollen, congested, and thickened condition of the terminal portion of the ileum and a regional adenopathy. In some cases the appendix was chronically inflamed, and in others it was acutely inflamed. Appendicectomy was the only operation performed in these cases. What has happened to such patients? Those whom we have been able to trace have not had any further symptoms, but will they in the future? This group of cases of early acute enteritis is not included in this study.

In the study of chronic types of regional enteritis, we have selected only the cases in which lesions originated in the small intestine and were not associated with true ulcerative colitis or with primary granuloma of the cecum. Wherever there has been doubt as to the presence of intestinal tuberculosis, even though the positive evidence was very scanty, we have omitted such a case from this study. Likewise, we have not included that small but most interesting group of solitary ileal ulcers and ulcers of Meckel's diverticulum which we[2] reported previously.

As our report is not only a clinicosurgical study of such cases but is especially concerned with a "follow up" study of the patients, we are including those cases previously reported by one of us (P. W. B.[1]). Adhering to our rigid selection, this report comprises 39 cases observed at the clinic from 1922 to date. The presence of the lesion was established by operation or necropsy. Thirty-six patients were subjected to operation at the clinic and two were

operated upon elsewhere. One patient died without being subjected to operation.

Clinical and Pathologic Features.—The ages of these patients ranged from nine to 61 years; one was ten years of age or less; three were between ten and 20 years; 11 were between 20 and 30 years; 14 were between 30 and 40 years; six were between 40 and 50 years; three were between 50 and 60 years, and one was 61 years of age. This age distribution parallels that found in a series of cases of ulcerative colitis, in which 29 of the 39 patients were less than 40 years of age. This probably is merely indicative that the

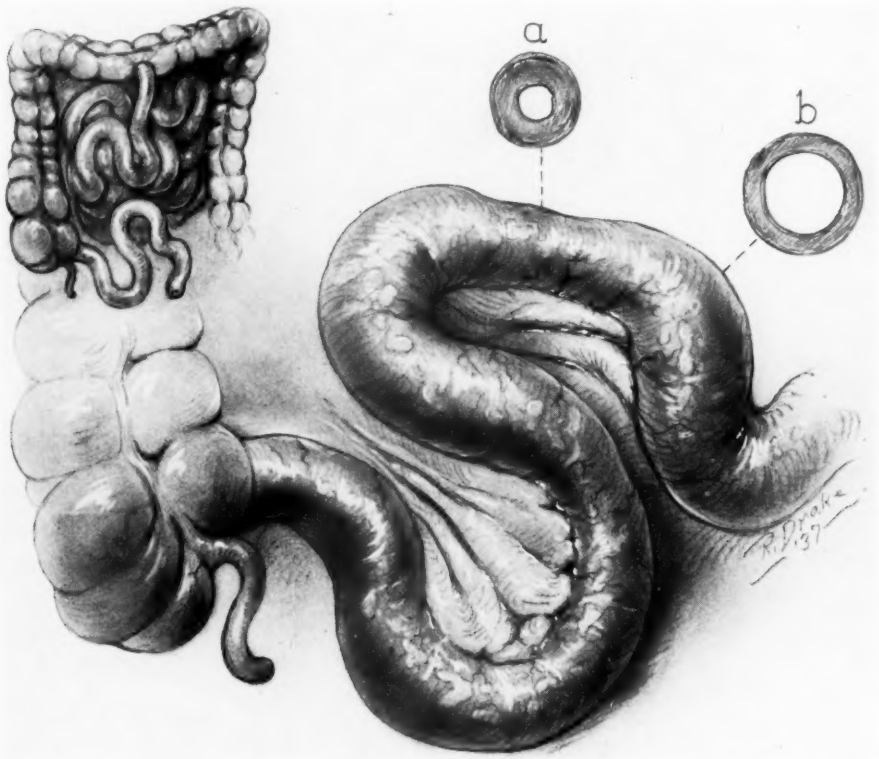

FIG. 1.—Active phase of ileitis involving terminal portion of the ileum; *a* and *b* represent the relative thickness of different segments of the ileum.

more active lymphoid tissue of young people is an important predisposing factor in any inflammatory disease. The sex factor was not significant in the cases of regional ileitis as 23 of the patients were males and 16 were females.

Situation of Lesion.—In the three cases in which the jejunum only was involved, the involvement was extensive. The ileum was involved in 34 cases. In 24 of these cases the involvement was in the lower or terminal portion of the ileum, in four cases it was in the upper part of the ileum, and in six cases it was in the terminal portion of the ileum and extended into the

cecum and ascending colon. Curiously enough, in these cases of ileocecal lesion, if the resection was carried out after a preliminary exclusion ileocolostomy, it was usually found that the disease was limited to the ileum and that the inflammation in the cecum had subsided. Finally, there were two cases of multiple involvement or "skip areas" throughout much of the small bowel. In these cases the narrowing was most marked in the lower part of the ileum.

Grossly the lesion consists of an inflammatory process which is rather sharply localized to a single segment of bowel but occasionally involves two or more segments that apparently are separated by intervening segments of normal bowel. In the more active phase the involved segment is greatly swollen, heavy, and reddened. It is commonly covered with plastic exudate and often is adherent to the brim of the pelvis and adjacent coils of intestines (Fig. 1). The mesentery attached to the diseased segment is stiff, enormously thickened, and contains enlarged hyperplastic lymph nodes. A slight amount of free turbid peritoneal fluid may be present. In the more chronic phase of the disease, which sometimes is seen at the primary operation and nearly always is seen when the abdomen is reopened following a previous short-circuiting anastomosis, the involved segment presents a different picture. At this stage marked edema, engorgement and exudate have to a large degree disappeared, but the intestinal wall still is greatly thickened. It feels leathery and in most instances is free of adhesions. Detailed pathologic study has failed to add to the original description of Crohn and his associates.

Clinical Picture.—In a few instances the initial symptoms are abdominal pain and diarrhea which last only a few days and do not differ from the symptoms which occur in many cases of acute enteritis, so-called intestinal flu, and which are self-limited. In other cases there are recurring attacks of pain and diarrhea which are not especially severe. In many cases the onset is insidious and is heralded by obstruction which produces acute pain, nausea and vomiting, and abdominal distention. In these respects, the disease parallels chronic ulcerative colitis.

Pain, which is the outstanding feature of the disease, was present in 38 of the 39 cases. Strangely enough, the one exception was a case in which there was extensive jejunal thickening. The chief symptoms in this case were nausea, weakness, and loss of weight. Efforts to localize the lesion by the distribution of the pain are helpful in only one respect; that is, the pain is more likely to be situated below the umbilicus than above it.

In 20 cases the salient features were attacks of pain which were associated with nausea, vomiting, abdominal fulness, and borborygmus, that is, signs of obstruction. In the 13 cases in which the mechanical obstruction was less marked, pain and diarrhea were the chief symptoms. In eight cases, a previous operation had been followed by a fistula which had been associated with persistence of pain.

As would be expected, fever, which often was associated with chills, occurred in 21 cases and no doubt occurred in others. Oddly enough, leukocytosis of any degree (more than 10,000 per cubic millimeter) was not common, although it was present in one case in which complete obstruction had been present for three days. In this case the leukocyte count was 19,000 per cubic millimeter of blood, and an emergency ileostomy gave evidence of peritonitis. Death ensued on the eighth postoperative day. Secondary anemia (value for the hemoglobin was 12 Gm. or less per 100 cc.) occurred in 17 cases. The lowest value for the hemoglobin was 8.5 Gm. per 100 cc. Anemia is not a prominent symptom and is related more to the duration of the disease and the deficiency of the diet than to actual loss of blood. This is contrary to what occurs in cases of ulcers of the ileum or ulcer of Meckel's diverticulum, in which hemorrhage is a common symptom. In one case in this series, tarry stools were associated with the attacks of pain and fever. The patient in this case was a boy, age nine, who had been ill four months. It is unusual that the boy has not had any symptoms since the ileocolostomy which was performed three years ago.

The patient who has regional ileitis may be of either sex and of any age, but usually is less than 40 years of age. The pain is most frequently situated in the lower half of the abdomen. The attacks usually have existed from four months to 20 years when the patient first seeks medical advice, the average duration being about four years. In this series of cases nausea and vomiting frequently were associated with the attack and in many instances they had led to an unsuccessful operation. Often, the real pathologic lesion had not been identified or an unfavorable prognosis of an inoperable malignancy or tuberculosis had been given. If the obstruction is not severe, diarrhea is a common accompaniment of the attacks of pain. There are no significant changes in the blood, although a macrocytosis is noted in some cases; but this can be considered only a suggestive sign.

The physical and laboratory data offer little help in the diagnosis. A tender mass, caused by an adherent inflamed loop of the terminal portion of the ileum, may be palpated, but this also is only suggestive. In the presence of any acute obstructing episode, the usual signs of obstruction would be observed. Abdominal fistulae are suggestive but not diagnostic until other data are at hand.

We are not the first to point out that a typical, pathognomonic clinical syndrome of regional enteritis has not been elaborated, that, in fact, the clinical diagnosis remains conjectural or tentative until roentgenologic evidence of the disease is adduced. Weber[1] comments on the roentgenologic diagnosis of regional enteritis as follows: "It may be assumed that the competently performed roentgenologic examination of the small intestine can be made to deliver diagnostic evidence of the disease at least as early as symptom-producing and sign-producing morphologic changes develop. Such evidence is almost entirely anatomic in character, reflecting not only the site, but also

the kind, intensity and extent of the pathologic changes. The early, relatively unpronounced degree of submucosal infiltration may be manifest roentgenologically only by flattening of the normally high mucosal relief pattern; if ulceration has taken place, the relief pattern is irregular and jagged, not smooth and flat. In more advanced stages of the disease the affected segment of intestine is shortened, its lumen is narrowed, and it loses its mobility and pliability,—all roentgenologically elicitable expressions of the productive inflammatory reaction of the disease. Associated with these anatomic changes are a relative hypermotility of the affected portion of the bowel and a relative hypomotility of the unaffected portions above and below it." That the serosal aspect of an affected loop of intestine may show little palpable or visible evidence of pathologic change was forcibly brought to our attention in a case in which the diagnosis of regional ileitis was made roentgenologically. Operation was not performed. The pathologist performing the necropsy found so little evidence of disease, from his examination of the serosal aspect of the small intestine, that he was inclined to view the roentgenologic diagnosis somewhat skeptically. Only after the intestinal wall and the mucosal surface were more intimately examined were the characteristic pathologic changes apparent.

Previous Operations.—The startling fact that 26 of these 39 patients had undergone one and often more unsuccessful operations for this disease is evidence of its seriousness, as is the fact that in the past the disease has remained unrecognized even after the abdomen has been opened. Sixteen patients in this series had undergone an appendicectomy which had failed to relieve their symptoms. Eight patients had fecal fistula which had followed previous operations, such as drainage of an abscess, or an effort to relieve obstruction in the terminal portion of the ileum by freeing the intestinal loops. In one case the fistula had developed after the ileum had been anastomosed to the ascending colon. A diagnosis of tuberculosis was made in a case in which the patient was subjected to a right salpingo-oophorectomy after an appendicectomy had been performed previously. In two other cases in which an exploratory laparotomy had been performed, the surgeon had made a diagnosis of tuberculosis and had closed the abdomen. The symptoms had recurred in four cases in which resection of the ileum or colon had been performed. Two patients who had been subjected to appendicectomy had been told that they had a disease of the ileum but they had not been advised about the proper treatment. A boy who had undergone an appendicectomy had been told that he had a disease of the ileum. One year after the appendicectomy had been performed, the boy had undergone an exploratory laparotomy. A diagnosis of lymphosarcoma had been made but no attempt had been made to remove the tumor. A fistula had developed in a case in which appendicectomy and drainage had been performed for either "cancer or actinomycosis."

Treatment.—The treatment of regional ileitis is essentially surgical and usually necessitates removal of the diseased segment with reestablishment of

the continuity of the intestinal tract. The operation may be performed in one or two stages. In a large proportion of the cases the disease is complicated by obstruction, by acute or subacute inflammatory changes, or by the presence of abscesses or intestinal fistulae when the patients are seen by a surgeon. It therefore is our belief that for reasons of safety the employment of the two stage procedure should be the rule rather than the exception. In cases in which the terminal portion of the ileum is involved, with or without extension into the cecum, it has been our practice to perform, as the first stage

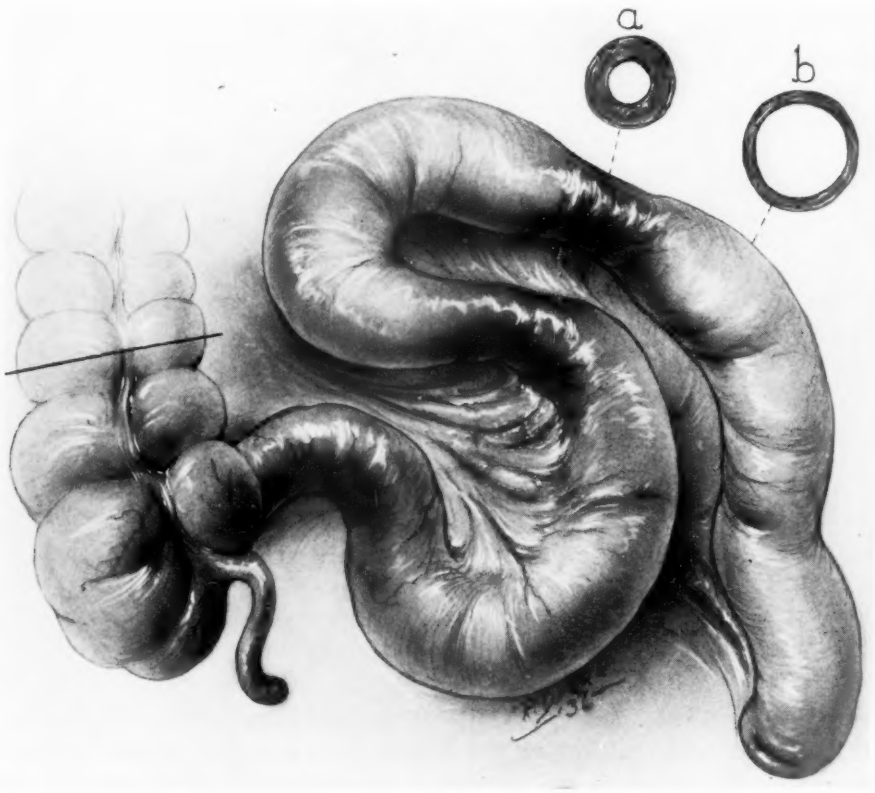

FIG. 2.—Subsidence of the inflammatory reaction; *a* and *b* represent the relative thickness of different segments of the ileum.

procedure, an ileocolostomy. A loop of ileum proximal to the lesion is united to the right half of the transverse colon, preferably as an end-to-side anastomosis. Since the principal purpose of this procedure is to exclude the diseased segment from the irritating effect of the intestinal current so as to permit partial subsidence of the inflammatory process (Fig. 2), it follows that a side-to-side ileocolostomy without division of the ileum distal to the anastomosis will not fully accomplish this purpose.

The value of an end-to-side ileocolostomy with complete diversion of the fecal stream, which permits subsidence of the inflammatory reaction in the

region of a fecal fistula and thus facilitates the subsequent resection, is illustrated by the following case.

Case Report.—A male, age 20, had an inflammatory lesion which involved the ileocecal segment. The lesion was complicated by a fecal fistula. Operation revealed that the mass was markedly fixed and surrounded by dense adhesions. A side-to-side anastomosis between the ileum and transverse colon was considered the safest procedure. Following this operation there was some improvement in the patient's health and some diminution in the amount of drainage from the fistulous tract. Two months later, resection was advised, but at operation the mass was still markedly fixed and the risk of resection was considered high. The ileum distal to the anastomosis was divided, thus completely excluding the ileocecal segment from the fecal current. There was almost complete cessation of fecal drainage, and the health of the patient improved markedly. At the time of resection, approximately five years later, the mass was quite free and the resection was readily done with minimal risk.

Although six of the eight patients who were subjected only to the first stage of the procedure (ileocolostomy) for localized enteritis reported that they were well and free of symptoms two to five years after operation, we believe that resection of the involved portion of the bowel is indicated in all cases in order to prevent the spread of the infection. In one case in which the patient delayed returning for the second stage (resection) for four years, a recurrence of the process was discovered in a short localized segment of ileum at the site of the previous end-to-side ileocolostomy.

We think that the interval between the stages of the procedure should be varied, depending chiefly on the general condition of the patient and the nature of the complicating lesion. In cases of localized ileitis which is not complicated by the formation of an abscess or fistula and in which the general condition of the patient is good, resection can be safely undertaken within three to four weeks after the initial procedure. In the cases in which the disease is associated with an enormously thickened mesentery, and in cases in which a fistula or an abscess has formed, there is a definite advantage in permitting two to four or even six months to intervene between the stages in order to permit partial resolution of the inflammatory process. In no instance have we seen any progress of the disease occur between the first and second stages when the interval did not exceed six months, but on the contrary there has been without exception a very marked subsidence of the inflammatory process which greatly facilitates resection.

Surgical treatment was employed in 36 cases. The operative procedure and the results are shown in Tables I, II, and III.

Table I reveals that extensive regional enteritis of the small intestine is a serious condition, and indicates that not much can be expected unless resection of the involved intestine is possible. In the two cases in which 70 cm. of the ileum were resected, the patients have remained well for one year, and for eight months, respectively. In the cases in which only short-circuiting procedures were possible, one patient is fairly well four years later, and the other three are little, if any, better. One patient died following an emergency ileostomy and two others died at home, months after the operation.

TABLE I

EXTENSIVE REGIONAL ENTERITIS

Patient	Site of Lesion (as Shown by Operation)	Surgical Procedure	Results
1	Entire jejunum	Exploration only	Died 7 months later
2	Upper portion of jejunum	Exploration only	Died 6 months later
3	200 cm. of jejunum	Gastrojejunostomy	Fairly well 4 years later
4	70 cm. of upper portion of ileum	Resection and anastomosis	Well 1 year later
5	Segment throughout small bowel (marked obstruction in lower portion of ileum)	Ileotransversostomy (partial exclusion operation)	Not well 22 months later
6	2 segments of midileum (acute obstruction)	Emergency ileostomy (general peritonitis present)	Died 8 days later
7	8 or 9 segments in lower portion of ileum (with obstruction)	Ileotransversostomy (partial exclusion operation)	Fair health 2 years later (associated pernicious anemia)
8	Multiple segments in lower portion of ileum, involving about 150 cm.	Ileotransversostomy (partial exclusion operation)*	Not well 7 months later
9	3 segments in lower portion of ileum	Resection of 70 cm. of lower portion of ileum and ileotransversostomy	Well 8 months later

SUMMARY OF RESULTS

Patients	Condition	Time after Operation
2	Well	(1 year) (8 months)
1	Fairly well	(4 years)
3	Not well	(2 years) (7 months) (2 years)
3	Dead	

* Resection later of terminal 150 cm. of ileum (December 2, 1936).

TABLE II

LOCALIZED REGIONAL ENTERITIS

Patients	Surgical Procedure	Results			
		Well	Not Well	Dead	No Data
1	Ileo-ileostomy	1			
5	Ileocolostomy	3	2		
2	Ileosigmoidostomy	1		1	
1	Closure of fistula and drainage			1	
2	Resection of lower portion of ileum and anastomosis	2			
5	Resection of lower portion of ileum and right colon with ileocolostomy (one stage)	2	2	1	
10	Ileocolostomy and later resection	9			1
1	Ileosigmoidostomy and later resection of lower portion of ileum	1			

In the 27 cases in which the process was localized in the lower part of the ileum (Tables II and III), results are more encouraging. In these 27 cases, we have further data on all but one patient. Nineteen patients are well, four are still having trouble, and three have died. In eight of the 27 cases, the short-circuiting operation was the only operative procedure employed. One of the patients has remained well for two years, one, for two and one-half years, and four, for from five up to six and two-thirds years with little, if any, further trouble. In one case in which drainage and an attempt at closure of a fistula were the operative procedures, the patient died at home, months later, of pulmonary complications. In 18 of the 27 cases, the diseased segment was resected. In four of these cases, the patients waited for one to five years before undergoing resection. The remaining 14 of the 18 patients underwent either a primary resection or the second stage of the procedure within two to four months after the initial operation. Of these 18 patients who had resection, two have continued to have further trouble characterized by persistence of fecal fistulae. One died following a one stage resection, and no recent data are available for one other patient. The remaining 14 patients are well. Eight of these patients have lived one year or less, one has lived for three years, and five have lived from four to 14 years since the operation. The last patient, who has now been in excellent health for more than 14 years since the resection, demonstrates that these patients should recover if the diseased segment is completely removed.

TABLE III

LOCALIZED REGIONAL ENTERITIS SUMMARY

Surgical Procedure	Total Patients	Results				No Data
		Well	Not Well		Dead	
Short-circuiting operation only	8	6 (1 for 2 yrs.; 1 for 2½ yrs.; 4 for 5 yrs. plus)	1	(same symptoms)	1 (2 mos. later at home)	
Resection of diseased segment	18	14 (8 for 1 yr. or less; 1 for 3 yrs.; 5 for 4 to 14 yrs.)	2	(fistula persists)	1 (6 days later)	1
Drainage and attempted closure	1				1 (8 mos. later at home)	

Data, therefore, are available in 35 of the 36 cases in which operation was performed. Twenty-two patients are apparently well, one is in fair health, and six are not well. Two deaths occurred in the hospital and four patients died after they returned home. The immediate surgical mortality was 5.5 per cent (two deaths). In these 36 cases, 47 major surgical procedures were carried out with a mortality of 4.2 per cent.

In three cases in which only a short-circuiting operation was performed, a deficiency syndrome with the hematologic picture of primary anemia has developed. It is impossible to say whether or not this syndrome is related

to the ileitis. All are being controlled by liver. In another case, a deficiency disturbance, comparable to the wet type of beriberi, developed after the operation. This disturbance cleared up promptly as a result of a normal diet, plus vitamin B.

SUMMARY

We have made a clinical study of 39 cases of regional enteritis and the surgical treatment and end-results in the 36 cases in which the patients were operated upon at the clinic.

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DISCUSSION.—DR. REGINALD H. JACKSON (Madison, Wis.).—I was greatly interested on Doctor Pemberton's timely paper on regional ileitis. Many surgeons have not, as yet, seen or recognized an instance of this distinct clinical entity. I should like to report three interesting cases of this condition which have come under my observation and which have presented some unusual features.

Case 1.—A girl, age 16, who, six months previous to admission, had had an appendicectomy performed elsewhere. Convalescence was unsatisfactory, the patient continuing to have intermittent attacks of acute abdominal cramps with tenderness in the right lower quadrant accompanied by nausea, vomiting, diarrhea and a rise of temperature to 101° F. The surgeon reported that at the operation he encountered a nest of adherent coils of intestine, and in digging out the appendix, opened a small, creamy abscess in the meso-appendix. The drainage had persisted for several weeks and the patient had developed a marked anemia and increasing lassitude. At operation the involved segment of the ileum and cecum was resected (Figs. 1, 2 and 3).

Such a history should immediately excite our suspicion of a terminal ileitis. As Doctor Pemberton emphasized, the actual diagnosis rests with the roentgenologist.

Whatever the etiologic agent is which is active in this disease, it would seem to be subject to recurrent periods of quiescence and activity covering many years, in some instances. This is brought out quite decidedly in the next case.

Case 2.—A female, age 30, who, in 1909, was operated upon for what was thought to be an interval appendix, having had, four weeks previously, an attack of acute abdominal cramps with vomiting, diarrhea and elevation of temperature to 101° F., at

FIG. 1.—(Case 1.) A telephotograph made during operation showing the typical thickening of the bowel wall of the terminal 18 inches of ileum and the enlarged lymph nodes in the mesentery, characteristic of this lesion.

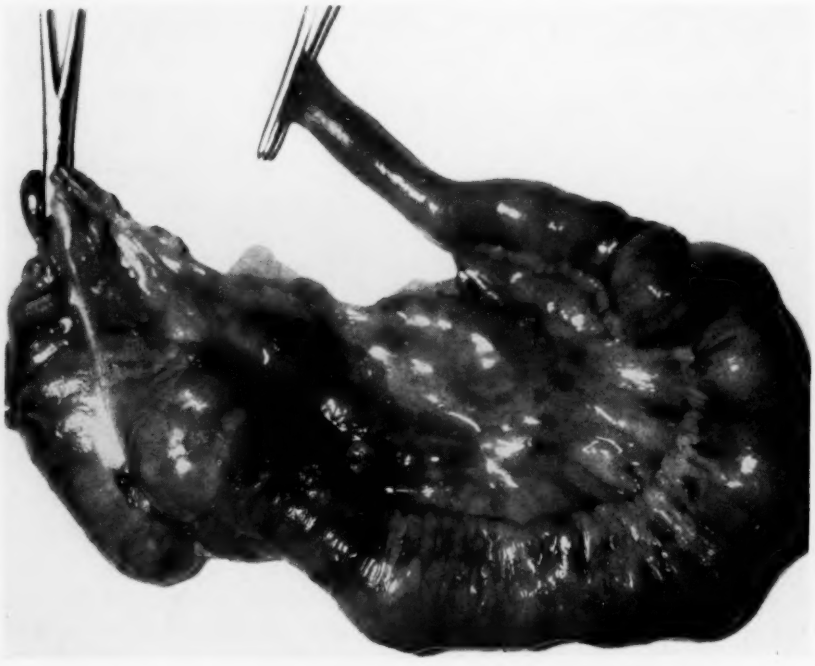

FIG. 2.—(Case 1.) Photograph of the specimen immediately after removal.

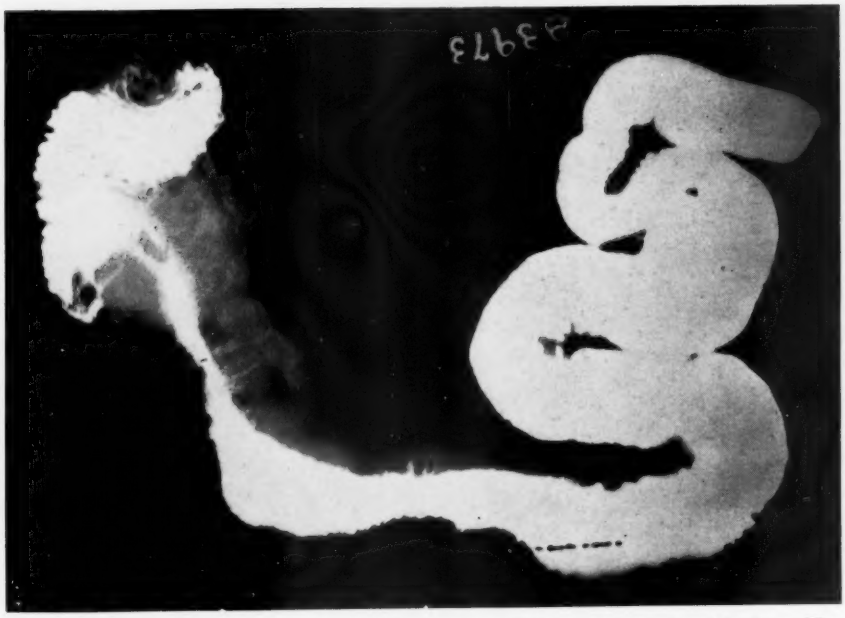

FIG. 3.—(Case 1.) Roentgenogram of specimen removed after it had been filled with barium. The so-called "string sign" is distinctly shown.

which time she was decidedly tender in the right lower quadrant. Exploration revealed some free serous fluid in the right lower quadrant, the terminal 18 inches of ileum enlarged, thickened, doughy and anemic—resembling a dead, water soaked night crawler.

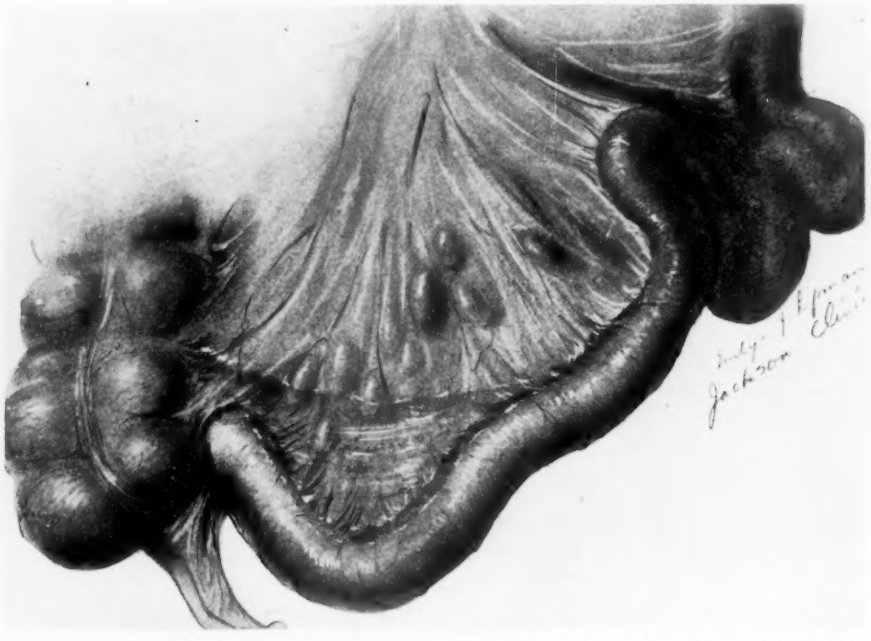

FIG. 4.—(Case 2.) Drawing showing the gross appearance of the pathology found at operation in 1909.

The attached mesentery had a dusky semithrombotic appearance and contained enlarged lymph nodes. The appendix was of the noncommittal type (Fig. 4). In view of the limited segment of the bowel involved and its appearance of favorable viability, resection

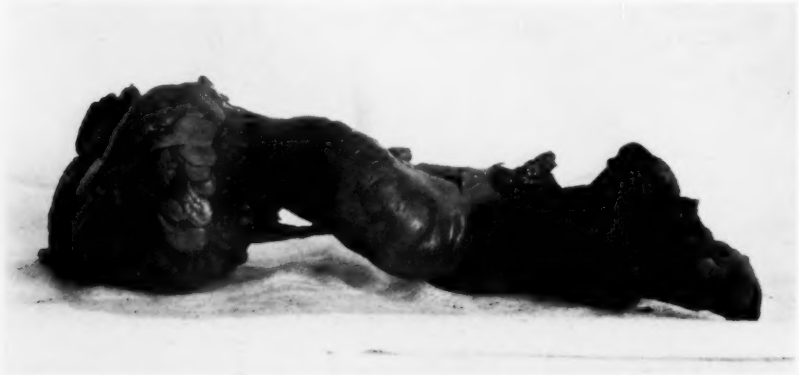

FIG. 5.—(Case 2.) Photograph of the fresh specimen of the segment of ileum removed in 1929. In its fresh state, it still retained a hose-like rigidity per se.

FIG. 6.—(Case 2.) Photograph of the specimen removed January 2, 1935. It consists of the terminal 28 inches of ileum, the cecum and part of the ascending colon, and exhibits the same hose-like rigidity in the fresh state as that noted in the previous specimen of ileum removed in 1929 (Fig. 5).

was not thought advisable; an appendicectomy was performed and the wound closed. I now believe that this was an early stage of a terminal ileitis.

For the next 12 years she was seen occasionally, and complained that she suffered intermittently from acute abdominal cramps, vomiting, diarrhea and pain in the right lower quadrant accompanied by a slight fever and malaise.

In 1922, the attacks became severe enough to require hypodermics of morphine. At this time a thorough clinical examination of the gastro-intestinal, biliary and urogenital systems was made with negative results.

From 1922 to 1929 she was fairly free from distressing attacks. Then they gradually increased in severity. Visible peristalsis was noted and a diagnosis of chronic intermittent intestinal obstruction was made and exploration advised.

At operation, in 1929, the involved terminal ileum, was resected (Fig. 5), after which the patient's general condition improved decidedly and she was free from symptoms until October, 1934, when the same cramping pain began to recur. At this time roentgenologic examination revealed a greatly diminished lumen of the terminal ileum which has been designated Kantor's "string sign."

FIG. 7.—(Case 3.) Drawing of gross pathology found at operation in the segment of upper jejunum, which resembled very closely that evidenced in the terminal ileum in Cases 1 and 2.

She was reoperated upon, January 2, 1935, and a practically exact reproduction of the condition found at the previous operation was revealed. There was no obstruction whatsoever at the point of the previous anastomosis nor did the disease invade the wall of the cecum (Fig. 6).

Professor Bunting noted that: "The lesion is like that of five years ago and is to me unique. There is a subacute or chronic inflammation involving mucosa, submucosa, muscular layer and subperitoneal tissues, many lymphoid and mononuclear cells and also fibroblasts, but strikingly many eosinophiles."

The patient has remained free from symptoms so far. This may not be a case of specific terminal ileitis under Crohn's definition, but it has been most interesting to follow its long course.

Case 3.—A male, age 57, who for several months had suffered from intermittent attacks of abdominal cramps accompanied by nausea, vomiting, slight fever to 101° F., and alternating spells of diarrhea and constipation, marked loss of weight, strength and a secondary anemia.

At operation pathology was found involving the proximal jejunum, practically duplicating that noted in the terminal ileum in Cases 1 and 2 (Fig. 7). The diseased segment of the jejunum was resected. Gross examination of the specimen showed the same type of thickening of the bowel wall as was noted in the previous cases with no definite point of obstruction, the disease progressing in the same tapering fashion as noted in Case 2.

The enlarged lymph nodes in these two cases suggests to me that the etiologic factor in some cases may be a fungus organism similar to Streptothrix. Professor Bunting has found spores in one instance but has, so far, not been able to develop cultures.

While I have some 15 records of resection of the cecal region for so called hyperplastic tuberculosis during the past 30 years, they seem to me, as I recall them, not to conform in the pathologic aspect or clinical course to this regional ileitis type first described by Crohn.

ENTERITIS OF THE OBSTRUCTED LOOP FOLLOWING ENTERO-ANASTOMOSIS FOR INTESTINAL OBSTRUCTION

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WHEN an intestinal obstruction is found to be due to a mass of adhesions involving the small intestine with no evidence of gangrene or strangulation, the operation of choice is often felt to be the establishment of an anastomosis around the obstructed and adherent coils. If the obstruction in the adherent loops persists, Holm and the author,[2] reported, in 1932, in both clinical cases and the experimental animal, that an ulcerative enteritis of these loops develops which is accompanied by a definite syndrome of diarrhea, vomiting, intermittent abdominal cramps, overactive peristalsis, loud borborygmus and dilatation of the involved intestine, which can be relieved only by complete resection of the entire side-tracked area. As our former report was based upon only two clinical cases and as in the last three years we have had three additional instances, it seemed of interest to review these cases in detail and to describe certain further observations that have been made in connection with this syndrome.

CASE REPORTS

Case 1.—Male, age 16. Admitted November 18, 1928, with symptoms and signs of acute intestinal obstruction of two days' duration. At operation several loops of the terminal ileum were found matted together and adherent to the lateral wall of the pelvis, around a gangrenous Meckel's diverticulum, causing a complete intestinal obstruction. The involved loops of ileum with the Meckel's diverticulum were resected, following which a lateral anastomosis was performed between the ascending colon and the ileum, four inches proximal to the resection. The four-inch blind limb of ileum distal to the anastomosis was placed over the large defect in the posterior parietal peritoneum of the pelvis where the intestinal coils had been adherent. Ten days after the operation the patient became slightly distended, and began having mild attacks of visible peristalsis in the lower abdomen which recurred during the remainder of his stay in the hospital. His general condition was good. After his discharge, December 7, 1928, the attacks of visible peristalsis recurred with increasing frequency and were associated with marked borborygmus and pain. He had from two to six loose bowel movements daily, the pain being relieved shortly before defecation. His symptoms becoming progressively worse, he was readmitted January 26, 1929, and was operated upon two days later. The four inches of blind ileum distal to the anastomosis, performed the previous November, had become greatly elongated and distended, and filled the lower half of the abdominal cavity, and was coiled upon itself, with dense adhesions to the cecum and pelvic wall. The blind end of the ileum was partially freed from adhesions when the patient's general condition became too serious to permit resection, and the operation was hastily terminated by performing a lateral ileo-ileostomy between a point near the end of the blind limb and an area immediately proximal to the former ileocolostomy. Recovery was prompt, and the patient was discharged February 9, 1929. Examination, September 21, 1929, showed the patient to be much underweight, due to a severe diarrhea of four months' duration. There had been occasional attacks of distention, borborygmus, and painful peristalsis. He was placed on a strict diet and by January 2, 1930, was greatly improved, although abdominal distention and a mild diarrhea had persisted. His appetite had been good throughout.

The patient was readmitted January 7, 1930, during an aggravation of the abdominal symptoms, and he was operated upon three days later. The lower two-thirds of the abdominal cavity were filled with the hugely elongated and dilated blind loop of terminal ileum, which was fully 18 inches in length and three to four inches in diameter and showed marked ulceration throughout. The mesenteric lymph nodes were all markedly enlarged. Proximal to the ileocolostomy the ileum seemed normal. The blind loop of ileum was freed of adhesions and resected and the sigmoid sutured over the denuded areas. Microscopic examination of the resected intestine showed a massive infiltration of small round cells into the mucosa and submucosa with some areas of destruction of the epithelial lining. This infiltration extended into the muscularis. There were secondary inflammatory changes with infiltration of fibrous tissue into the submucosa and muscularis. This was evidently a chronic inflammatory process that was still active in spite of the attempts at healing. The patient was discharged January 28, greatly improved, although he still had a mild diarrhea. The diarrhea persisted for several weeks after his return home. Fifteen months later, he was in excellent health, had gained 35 pounds in weight and had no gastro-intestinal complaint. He has remained well six years.

Summary.—A boy of 16, after resection of the terminal ileum for intestinal obstruction due to a Meckel's diverticulum, and lateral ileocolostomy, with a short loop of blind ileum left distal to the anastomosis, developed a severe enterocolitis with low abdominal distention, pain, and borborygmus, and was only slightly relieved until a complete resection of the blind loop was finally performed, the loop having become hugely elongated, distended, and ulcerated, filling almost one-third of the abdomen. He has remained well. This case has been cited because it presents a clinical picture quite similar to those following, due to the ulceration and dilatation of the short blind end of ileum—beyond the anastomosis—which had been allowed to remain, as it was feared that postoperative adhesions of the small intestine might again produce obstruction. It was deliberately planned to use this blind end to cover over the raw area and to meet any complications that might result later from this procedure.

Case 2.—Male, age 12. Admitted June 11, 1929. Three days before admission, the patient was seized with acute appendicitis, and at operation one hour after admission a gangrenous, perforated appendix was found within a large abscess walled off by intestinal coils. Appendicectomy was performed and the abscess drained. Convalescence was prolonged because of wound infection, but otherwise was uneventful. At a follow up examination October 17, 1929, the patient was in excellent health. He remained free from abdominal symptoms until April 5, 1930, when intestinal obstruction occurred. At operation, the terminal three feet of ileum were found adherent to themselves, to the cecum, and to the anterior abdominal wall. Adhesions were divided to release a three inch loop of strangulated but viable ileum. Inasmuch as extensive, dense adhesions threatened future strangulation, a lateral anastomosis was established between the transverse colon and the ileum at a point proximal to the adhesions. Throughout his convalescence the patient had a good appetite and his general condition was excellent. A mild, painless diarrhea, with considerable borborygmus and moderate distention of the lower abdomen, developed one week before his discharge on April 22, continued in a mild form until May 4, when the diarrhea became severe and painful, and vomiting occurred. These symptoms became progressively worse, and on readmission, May 9, the patient was undernourished, dehydrated, drowsy, and very toxic. The abdomen was moderately distended and generally tender, but there was neither rigidity nor palpable mass. At operation the following morning, the terminal three feet of ileum, side-tracked on April 7, were found elongated to seven feet, greatly dilated, acutely inflamed and ulcerated,

with the distal one foot completely obstructed in a mass of adhesions. The mesoileum contained many lymph nodes varying in size from that of a pea to a walnut. The colon was collapsed proximal and distal to the anastomosis. The side-tracked loop was resected from the anastomosis to the cecum. During the manipulations of resection, several ulcerations of the loop perforated. The abdominal cavity was drained. A serious wound infection and the preoperative debility prolonged the convalescence. A mild diarrhea persisted throughout the patient's stay in the hospital and continued for several months after his discharge. Recuperation was slow for several months following his return home, but at the follow up examination one year later, May, 1931, he had gained 25 pounds in weight and his general health was excellent. He still is quite well, five years later.

Summary.—A boy, age 12, nine months after an appendicectomy and drainage of an appendiceal abscess, developed an intestinal obstruction due to massive postoperative adhesions of the ileum which was relieved by an ileocolostomy around the obstructing coils. Postoperative diarrhea supervened with low abdominal pain, distension and borborygmus which failed to respond to treatment. One month later reoperation became necessary. The terminal side-tracked ileum was found to be a hugely distended mass of adherent intestinal coils, ulcerated, acutely inflamed, and obstructed. Following resection of this ulcerated and obstructed mass, the patient made a slow but satisfactory recovery and has remained well ever since.

Case 3.—A woman, age 45, was admitted July 6, 1934. She had had, in 1914, a bilateral oophorosalpingectomy and appendicectomy, tonsillectomy, in 1920, hemorrhoidectomy, in 1922, and an operation for acute intestinal obstruction, in 1925, with release of omental adhesions. In 1930, there was again acute obstruction of the ileum due to a mass of adhesions of the ileum, sigmoid, omentum and transverse colon, and an ileocolostomy was performed, from a point above the obstruction to the middle of the ascending colon. Recovery uneventful. Wassermann four plus.

Following this operation, at intervals of three to four months, the patient had peculiar attacks of severe generalized abdominal pain with diarrhea followed by the expulsion of a very large mass of loose feces. Between attacks, cathartics were required. Some relief was obtained by colonic irrigations. On admission, the left lower quadrant of the abdomen was prominent and distended, and gurgling peristalsis was audible without a stethoscope (borborygmus).

At operation the terminal ileum was found to be involved in a mass of firm adhesions and was matted together with the omentum, cecum, sigmoid, and adherent to the abdominal wall. The former anastomosis was found to be from the middle of the ileum to the ascending colon. There was no complete obstruction found in the side-tracked loop but numerous constricted areas and an occasional small superficial ulcer were noted. The regional mesenteric lymph nodes were enlarged. The adhesions involving this loop were separated, and it was resected down to the ileocecal valve. Convalescence was uneventful except for a postoperative diarrhea that subsided within a week.

Follow Up Report.—Two years and four months after the operation, patient had had no further attacks of diarrhea, and constipation was relieved. She still had headache and backache, probably due to lues.

Summary.—A woman, age 45, who, following an ileocolostomy for intestinal obstruction due to a mass of adhesions involving the terminal ileum, developed attacks of generalized abdominal pain, diarrhea and borborygmus. She was, apparently, completely cured by a resection of the side-tracked ileum, which showed persistent constrictions due to the adhesions, no complete obstruction but a mild ulcerative enteritis.

Case 4.—A man, age 29, was admitted, June 9, 1932, with symptoms of acute intestinal obstruction. In 1915, appendicectomy had been performed elsewhere. In 1928, an acute intestinal obstruction had been relieved by simple release of adhesions. One year and six months later, he had symptoms of obstruction with spontaneous recovery. Operation revealed a mass of dense adhesions that seemed to involve almost the entire ileum. A few obstructing adhesions were released and an ileocolostomy performed from a point proximal to the adhesions to the middle of the transverse colon. Convalescence uneventful. He was discharged in two weeks, having no further pain, but averaging four loose stools a day. This diarrhea never ceased. Five months after discharge he began to have gurgling sounds over the right lower abdomen and he could feel gas or fluid passing a point in that region accompanied by a sharp pain; these sensations were aggravated by eating or drinking. He passed large amounts of gas and liquid stools that at times were difficult to control. He had vomited intermittently for weeks, had lost 20 pounds in weight and had become quite weak. He was readmitted February 2, 1933. There were fulness in the lower abdomen, almost hourly intervals of borborygmus, sharp crampy pain, and three to four loose stools a day. Roentgenologic examination showed stasis in the small intestine situated in the right lower abdomen. Operation, Febraury 6, 1933. The right lower quadrant of the abdomen was filled with a mass of adherent small intestines which were found to be the previously obstructed lower portion of the ileum. There was still a partial obstruction in the side-tracked loop about 40 cm. from the previous ileocolostomy, due to the dense adhesions which narrowed the lumen of the gut to about one-quarter of its usual diameter. There was also a mass of very large, nonsuppurating mesenteric lymph nodes present. This portion of the ileum was quite friable, and on separating the adhesions, the serosa was torn. Numerous ulcers of the mucosa were found just proximal to the obstruction. The entire side-tracked portion of the ileum was gently freed and resected down to the cecum. The cecum and ascending colon were not resected. Convalescence was uneventful except for the diarrhea, which persisted for almost three months following operation.

Follow Up Report.—Four years and nine months after operation the patient was completely well. No further pain, diarrhea or indigestion. He had gained 25 pounds in weight.

Summary.—A man, age 29, following an appendicectomy, developed repeated attacks of acute intestinal obstruction which finally required an ileocolostomy around the obstructing ileal adhesions. Diarrhea, abdominal pain, with loud gurgling, vomiting and loss of weight ensued. The condition was completely relieved eight months later by resection of the side-tracked loops of ileum, which showed an ulcerative colitis proximal to a persisting stenosis caused by the dense adhesions.

Case 5.—A female, age 30, was admitted August 5, 1936. Nine years previously she had had a bilateral oophorosalpingectomy and an hysterectomy performed elsewhere for, apparently, a severe pelvic inflammatory condition. Ever since, patient has been exceedingly nervous accompanied by an almost continuous series of hysterical manifestations.

In July, 1934, she had had a cholecystectomy for chronic cholecystitis. In January, 1935, she was readmitted with symptoms of acute intestinal obstruction. At operation no acute obstruction was found, but there were adhesions of the ileum 90 to 100 cm. from the ileocecal valve, and a coil of intestine which had become caught beneath adhesions of the mesentery of the ileum to that of the cecum caused a kinking and partial obstruction. Simple freeing of the constricting adhesions did not promise complete relief. An anastomosis, therefore, was effected between a loop of ileum above and one below the adherent area. Convalescence was uneventful. She remained comparatively well for nine months. Ten months before readmission she began to have mild attacks of diarrhea and vomiting with severe epigastric pain radiating to the back. At first the attack

would last four to five days and recur only once in four to six weeks, but the last attack has persisted for four weeks with practically continuous diarrhea and frequent vomiting. On admission, by the use of a Wangensteen suction drainage tube, she was relieved for 48 hours, but the diarrhea—thin greenish stools—again began with sharp abdominal pain, synchronous with borborygmus, but no visible peristalsis. Roentgenologic examination with a barium enema showed a patent ileocecal valve with dilated coils of terminal ileum (side-tracked coils). Exploration showed the old side-tracked loop adherent in coils, with no definite obstruction except that one adhesion to the cecum seemed to narrow the lumen. There were no enlarged mesenteric lymph nodes but there were a few small ulcers of the mucosa 5 to 10 cm. proximal to the constriction. The involved side-tracked area was resected. There was no postoperative diarrhea. Convalescence normal.

Microscopic examination of the resected loop showed a small round cell infiltration involving all coats of the intestine, being most prominent in the mucosa, submucosa and serosa. In some areas polymorphonuclears; in others lymphocytes predominated. There was a scattering of eosinophiles. There was a definite perivascular round cell infiltration of the serosa. Diagnosis: Chronic interstitial ileitis.

Follow Up Report.—Four months after operation. No further intestinal symptoms. No abdominal pain. Eating well. No indigestion, but has tremor of the left upper extremity and is bedridden (hysteria).

Summary.—A woman, age 30, who, nine months after an ileo-ileostomy for intestinal obstruction due to intestinal adhesions, began to have attacks of diarrhea, vomiting, and abdominal pain, gradually becoming very severe and continuous; relieved by resection of the side-tracked ileum which showed a slight constricted area, and in which there were present an enteritis and a few mucosal ulcers.

COMMENT.—Four cases are herewith recorded in which, following an entero-anastomosis to relieve an intestinal obstruction, due to massive adhesions, an enteritis of the obstructed loops developed, as evidenced by recurrent attacks of diarrhea, abdominal pain, overactive peristalsis, borborygmus, and at times vomiting, which were entirely relieved by resection of the side-tracked loops. In these loops there were apparent stasis, some persistence of the obstruction or constriction of the gut, enteritis and ulceration. The ulceration was more evident, the more nearly the obstruction remained complete. This syndrome was quite similar to that appearing in a fifth case in which a long distal blind loop was permitted to remain beyond a lateral anastomosis, which was also cured by resection of the tremendously dilated blind loop.

Experimentally, we have demonstrated that an enteritis with ulceration can be produced in dogs by an anastomosis around an intestinal obstruction whether the anastomosis be an ileo-ileostomy or an ileocolostomy. Furthermore, in the experimental animal the enteritis may be found to extend into the ileum proximal and into the colon distal to the anastomosis. It, therefore, seems fair to assume that a similar condition may exist in the human and is responsible for the diarrhea that in some instances persists after resection of the involved loops.

Pearse[5] has recently reported experimental work similar to ours. He has shown in dogs that:

(1) When a long blind loop is left proximal to an anastomosis for intestinal obstruction, peristalsis is away from the obstructed blind end, the loop empties easily, the dogs remain well.

(2) When a blind loop is left distal to an anastomosis, the peristalsis is toward the obstruction or blind end, and he believes that:

(*a*) Short jejunal loops up to one foot in length are tolerated well and empty themselves.

(*b*) Loops from three to four feet long become filled with inspissated feces and débris and dilate enormously; if perforation does not occur, emaciation, anorexia, lassitude, and lethary ensue and finally death.

(*c*) Loops five to six feet long cause death from inanition and dehydration. In six dogs, as in two of ours, death occurred from peritonitis following perforation of an ulcer. Other superficial ulcerations were occasionally observed, but he believed they were due to pressure necrosis from bits of bone and foreign bodies that collect in these blind loops.

He also described a clinical case which, six months after a lateral anastomosis around an obstruction, developed attacks, at intervals, of abdominal discomfort, anorexia, nausea, vomiting, and loss of weight. Resection of the side-tracked segment, which was thick, dilated and ulcerated, was followed by diarrhea for three weeks, after which the patient remained well.

Pickhardt[6] refers to a right hemicolectomy and ileocolostomy which nine years later showed a dilated and elongated blind end of the ileum unaccompanied by symptoms. Stettin in discussing this case mentions the perforation of a similar blind loop.

Vaughan,[7] in arguing for entero-anastomosis as against enterostomy in ileus, cited one case in which "symptoms were much improved by entero-anastomosis," but resection of the side-tracked intestine was necessary for complete relief; in another case, after anastomosis, recurring attacks of abdominal pain and vomiting persisted and resection of the adherent coils of small intestine was necessary in which "many obstructive points were encountered forming dilated bowel areas filled with fecal matter and pus." Three other of his cases remained well without resection.

Little, Zerfos, and Trusler[4] report a case in which an anastomosis between the jejunum and the ascending colon and jejuno-ileostomy had been performed for intestinal obstruction. The patient had marked loss of weight with periodic abdominal cramps, vomiting, diarrhea, marked enemia, and visible peristalsis. The entire small bowel between the first anastomosis and cecum was markedly distended, thickened, and filled with liquid bowel contents. Both anastomoses were taken down and the piece of jejunum between the openings was resected with end-to-end union. The excised bowel showed marked hypertrophy of the muscle, edema and hemorrhage. The abdominal symptoms were relieved but a primary type of anemia with fatigue, anorexia, and loss of weight persisted, which, however, responded to liver extract and the usual pernicious anemia therapy.

Christopher[1] describes a similar case in which after a jejunocolostomy for obstruction, meteorism, flatulence, belching, tremendous appetite, marked pallor and anemia developed. The entire small intestine was enormously distended and the walls thickened, but there was no evidence of ulceration. The

terminal ileum was markedly adherent. The jejunocolostomy was taken down and an ileocolostomy around the adherent coils of ileum was followed by rapid recovery. Urmy, Ragle, Allen and Jones[8] have reported a case of beriberi following short circuiting of the small intestine.

Apparently, therefore, both Vaughan and Pearse have observed cases presenting symptoms similar to ours, which have been cured by resection of the side-tracked intestine. Also, Christopher and Little, Zerfos, and Trusler's experiences with very long side-tracked loops of small intestine, as well as Pearse's experiments with dogs, indicate that an entero-anastomosis, short circuiting long lengths of small intestine, may lead to grave anemia and serious toxemia.

CONCLUSIONS

(1) Entero-anastomosis for intestinal obstruction due to massive adhesions may be a very useful procedure but it may have certain limitations and complications.

(2) Following an anastomosis around a mass of intestinal coils obstructed by adhesions, if the obstruction persists, there will follow an ulcerative enteritis of the obstructed loops with perhaps a general enterocolitis, as evidenced by diarrhea, abdominal pain and borborygmus. Resection of the side-tracked intestine will be necessary. Even stenosis or a persistent partial obstruction may lead to a similar syndrome.

(3) Entero-anastomosis for intestinal obstruction must be looked upon as a possible first stage operation of which resection of the obstructed loops may be required at a second stage.

(4) In the presence of acute obstruction it would be unwise to consider primary resection of the obstructing loops, but if at the time of operation it seems likely that the obstruction will remain, the two stage operation should be definitely planned.

(5) It is further suggested that, as an alternative procedure to a lateral anastomosis around the obstruction, the intestine might be divided as close to the obstructive lesion as possible, followed by an end-to-side anastomosis below the obstruction. In this manner a proximal instead of a distal blind end would be left which should drain normally, give no symptoms, and require no further operative procedure.

(6) Short circuiting very long lengths of small intestine may lead to serious toxemia, grave anemia, or a syndrome similar to beriberi.

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A CONTROLLABLE CECOSTOMY

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CECOSTOMY is perhaps the most advantageous method of decompressing an obstructed colon. It may be used as a preliminary to a subsequent resection of the colon, or as a safety valve in cases in which a resection of part of the left colon is followed by primary anastomosis. It is usually a temporary procedure but may have to serve as a permanent opening. Rankin urges that no exploration be carried out in cases of acute obstruction from a malignancy at the time cecostomy is performed. In a certain number of such cases at the time of the second operation, the growth is found to be inoperable, or there is metastatic involvement of the liver. In these cases the cecostomy is left as a permanent relief for the obstruction.

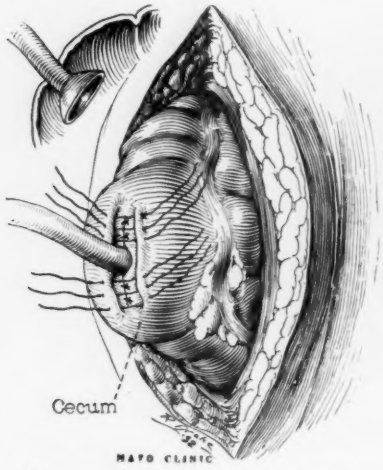

FIG. 1.—Showing the technic of using a de Pezzer catheter in performing cecostomy. (From "The Colon, Rectum and Anus," Rankin, Bargen, Buie, W. B. Saunders.)

FIG. 2.—A piece of monel metal approximately ten centimeters square on which has been soldered a climax clamp (A). One handle is left free, so that pressure at (C) will open the clamp, and the end of catheter is brought through (B) while the clamp is open.

Whether temporary or permanent, it is highly desirable that the drainage from the colon be controlled. It not only makes the patient much more comfortable and happier, but also makes the probability of contamination at the second operation much less likely.

No originality is claimed for the method of performing the cecostomy. A large de Pezzer catheter is used after having cut off the top of the mushroom end. The cecum is delivered through a McBurney incision and a rubber covered clamp applied. The catheter is then introduced into the cecum in the manner described by Gibson (Fig. 1), as modified by Hendon and Rankin. A purse string suture is placed in its wall and the cecum opened, the catheter

introduced and suture tied. Reinforcing sutures or a second purse string is inserted and the cecum is attached to the parietal peritoneum. The cecal or colonic contents thus drain through this catheter, which in turn may be connected by a glass tube to a long rubber tube and drained into a bottle. This works well as long as the patient is in bed. There is practically no contamina-

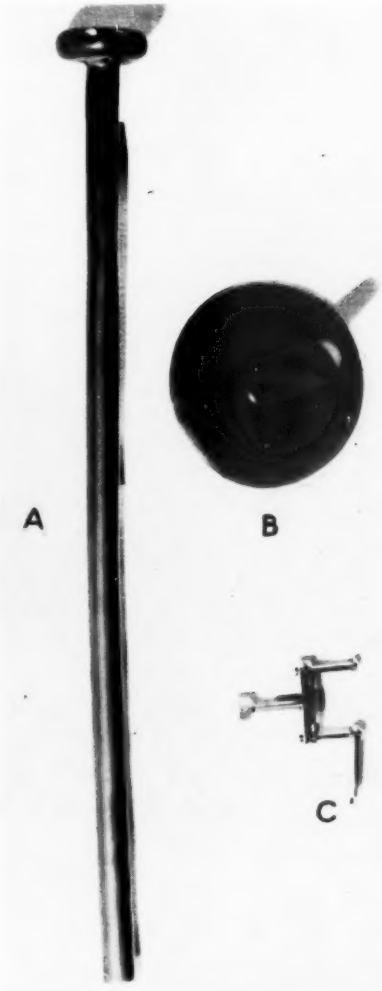

FIG. 3.—(A) Open end de Pezzer catheter. (B) Rubber retention disk. (C) Clamp from clysis set.

FIG. 4.—Parts shown in Fig. 3 assembled.

tion of the dressings. When the second stage is attempted, if metastases are found in the liver, the cecostomy is left a permanent one.

In such cases, or where the second stage is not performed immediately, it is desirable for the patient to get out of bed. The tube then has to be removed and fecal contamination is not controlled. In an attempt to control this, a hole (Fig. 2B) exactly the size of the catheter was drilled in a piece

of monel metal ten centimeters square, and a climax clamp (Fig. 2A) was soldered on one side. The end of the catheter was put through the opening and some gauze placed between the metal and the patient's skin. The metal plate was held in place by adhesive strips and more dressings applied over it. Each morning the clamp was opened (by pressure at Fig. 2C) and a colon irrigation given. At intervals during the day the clamp was released to allow the patient to pass feces.

The patient was able to get out of bed and live as normal a life as her strength would permit. At first it was thought that the catheter would probably work loose and come out. It did not. In the first case in which it was used, after being in place for many months, the catheter was removed, because of irritation of the edges of the wound. The patient was confined to her bed at this time and the tube was never replaced. A copper tube, about five inches long and slightly larger than the catheter, had been secured to aid in replacing it if necessary. The mushroom end was to be pulled into the metal tube, which in turn was to be introduced into the cecum through the well established sinus. Then the catheter could be pushed out of the tube into the colon.

It was felt that a rubber protecting disk would be more comfortable than a metal plate, and such a "retention disk" (Fig. 3B) was found. It is usually employed over a de Pezzer catheter after suprapubic cystostomies. This apparently would work over a cecostomy tube also. One was tried and was held in place either with liquid adhesive or strips of adhesive plaster and proved much more comfortable. A clamp from a clysis set (Fig. 3) is used to close the tube and can be easily managed by the patient. Instead of cutting off the top of a catheter, an open end de Pezzer catheter (Fig. 3A) can be obtained and is more satisfactory (Figs. 3 and 4).

By the simple means of using a metal or rubber shield, and a clamp, the fecal discharge of a cecostomy can be controlled, thus preventing the contamination of the field if a second operation is needed, and making the patient much happier and more comfortable if the cecostomy has to be left as a permanent one.

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1833 Pine Street, Philadelphia, Pa.

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Subscriptions, advertising and all business communications should be addressed

ANNALS OF SURGERY
227 South Sixth Street, Philadelphia, Pa.